Hypothermia

Cold Injuries and Cold Water Near Drowning

Socialstyrelsen
THE NATIONAL BOARD OF HEALTH AND WELFARE

Dedication

This second edition is dedicated with thanks to Dr William J. Mills Jr., in recognition of his clinical contributions that have changed the treatment of frostbite and hypothermia around the world.

ISBN 91-7201-640-X
Article no. 2002-110-14

Typesetting: Fhebe Hjälm
Cover photo: Åmsele -35 °C, Helge Brändström
Printed by Modin-Tryck, Stockholm, 2002

Preface to Second Edition

Since the first edition of this report, written in 1996, several important changes have taken place in the management of patients with hypothermia and local cold injuries. This edition, therefore, has been produced in response to these changes.

The revision has been made by an Editorial Committee consisting of Dr *Helge Brändström*, Senior Consultant of Anaesthesiology, Umeå University Hospital, Sweden, Dr *Bruce C. Paton*, Clinical Professor of Surgery, University of Colorado School of Medicine, Denver CO, USA and Dr *Murray Hamlet*, formerly of the US Army Research Institute of Environmental Medicine, Natick MA, USA.

The section written by *Ingvar Holmer*, Professor of Climate Physiology, National Institute of Working Life, Stockholm, Sweden has been revised and updated by the author.

Two new sections have been added: "Near Drowning" contributed by Dr Hamlet, and "Avalanches" contributed by Dr *Bruno Durrer*, Lauterbrunnen, Switzerland and Dr *Hermann Brugger*, Bruneck, Italy.

The report was reviewed by Dr *Ulf Björnstig*, Clinical Professor of Surgery, Umeå University Hospital, Sweden, Dr *Karl-Axel Ängquist*, Clinical Professor of Surgery, Umeå University Hospital, Sweden, Dr *Per-Ola Granberg*, Clinical Professor of Surgery, Karolinska Hospital, Stockholm, Sweden, Dr *Daniel Danzl*, Professor and Chairman, Department of Emergency Medicine, University of Louisville, Louisville, KY, USA, and Dr *Gordon Giesbrecht*, Professor of Physiology, University of Manitoba, Winnipeg, Canada.

The Editors record with regret the death of Dr *Börje Renström*, who contributed to the first edition, and died during the preparation of the second edition.

We would like to give special thanks to *Karin Hedlund* who gave many hours of invaluable help editing and typing the manuscript.

Bo Lindblom

Preface to the First Edition

The variegated geography of Sweden, with its combination of mountain ranges, a long coast line, many lakes, long distances and a low ambient temperature for much of the year, entail risks of hypothermia.

Swedish medical personnel need to know more about hypothermia, its causes and treatment. This in turn demands both good and readily available factual data and the inclusion of the subject in training and exercises.

The National Board of Health and Welfare has therefore compiled this report on Hypothermia. The report is intended for training use and as practical help for medical personnel.

The report has been compiled, on the board's behalf, by *Helge Brändström*, Deputy Senior Consultant (Anaesthesiology Department), and Ass. Prof. *Ulf Björnstig*, Surgical Department, both of the Umea University Hospital.

Work on the report has proceeded on a project basis, and the project steering group was chaired by *Karl-Axel Norberg*, Head of Division at the National Board of Health and Welfare. Others taking part in the project were Professor *Sten Lennquist*, Dr *Börje Renström* and Principal Administrative Officer *Ulla Wistrand*.

Professor *Per-Ola Granberg*, Professor Ingvar Holmér and *Anders Holmström* M.D. Ph.D. served as referees.

As work progressed, Professor *Daniel F. Danzl* (University of Louisville), Director *Murray P. Hamlet* (U.S. Army Research Institute of Environmental Medicine) and Rear Admiral, Dr *William J. Mills* (Anchorage) contributed knowledge, research data and experience pertaining to the practical treatment of hypothermia cases. The report was also reviewed by Director *Franklin R. Hubbell* D.O (SOLO, Conway, New Hampshire). Appendices 2, 4 and 5 to the report were written by Dr *B. Renström*, App. 3 by Professor *I. Holmér*.

Training material is to be published as an adjunct to this report.

Claes Örtendahl

Contents

How many degrees?

The temperatures mentioned refer to the Celsius system.
The equivalent in Fahrenheit is shown in the left-hand column.

Fig 1. Fahrenheit to Centigrade Conversion Scale*

Fahrenheit	Centigrade	Fahrenheit	Centigrade
95	**35**	63	17.22
94	34.44	62	16.67
93	33.89	61	16.11
92	33.33	60	15.56
91	32.78	**59**	**15**
90	32.22	58	14.44
89	31.67	57	13.89
88	31.11	56	13.33
87	30.56	55	12.78
86	**30**	54	12.22
85	29.44	53	11.67
84	28.89	52	11.11
83	28.33	51	10.56
82	27.78	**50**	**10**
81	27.22	49	9.44
80	26.67	48	8.89
79	26.11	47	8.33
78	25.56	46	7.78
77	**25**	45	7.22
76	24.44	44	6.67
75	23.89	43	6.11
74	23.33	42	5.56
73	22.78	**41**	**5**
72	22.22	40	4.44
71	21.67	39	3.89
70	21.11	38	3.33
69	20.56	37	2.78
68	**20**	36	2.22
67	19.44	35	1.67
66	18.89	34	1.11
65	18.33	33	0.56
64	17.78	**32**	**0**

* C = (F -32) x 5/9. Each 5 °C = 9 °F.

Introduction

Accidental hypothermia is defined as unplanned reduction of body temperature to below 35 °C. The history, epidemiology, pathophysiology and treatment of hypothermia are discussed

Man's ability to maintain a constant core body temperature (37 °C $^{+}$0.5 °C) enables him to dwell and survive at various temperatures. When the ambient temperature is approximately 27 °C (thermoneutrality without clothing is 29 °C), a person is in a state of thermoneutrality, which means that at rest and without clothing we maintain normal body temperature without requiring insulation, heat through shivering or from exogenous heat sources. At ambient temperatures between 15 °C and 60 °C we can maintain a constant core temperature without clothing, so long as the air about us is dry and so long as the body's thermoregulatory mechanisms are functioning. When the ambient temperature falls below 15 °C, we require greater physical activity and need protective clothing in order to stay warm. A lower ambient temperature makes us seek shelter, increase our food intake and augment our muscular exertion.

Man is equipped with both peripheral and central thermoreceptors for receiving signals to regulate body temperature. The peripheral receptors, most of which are in the skin, detect changes in the ambient temperature. The central receptors detect temperature changes in the blood and the core of the body.

Central and peripheral signals travel, along neural pathways in the spinal cord, to a temperature regualtion control centre in the hypothalamus where an integrated thermal signal is determined.

When our skin becomes cold or body temperature falls below 37 °C, thermoregulatory mechanisms are activated to decrease heat loss (i.e. peripheral vasoconstriction)and increase heat production (i.e. shivering).

Hypothermia During body cooling means that:

- at a body core temperature between 32 to 35 °C (mild hypothermia), thermoregulatory mechanisms are able to function maximally in an effort to prevent or slow down further body cooling.
- at a body core temperature of between 28 to 32 °C (moderate hypothermia), thermo-regulatory mechanisms are not functionally fully and actually start to fail,
- at a body core temperature of less than 28 °C (severe hypothermia), thermo-regulation is virtually eliminated,
- at a body core temperature below 26 °C, the body is poikilothermic, eventually assuming the temperature of its surroundings.

In a healthy individual, shivering alone can increase heat production two to five-fold. Shivering is an unsynchronised high-frequency muscular contraction of agonists and antagonists, combined with a slow, synchronised, centrally triggered muscular contraction, the intensity of which varies over time. Conversely, if core temperature rises, heat emission can be increased ten-fold by evaporation and perspiration.

Any injury or pathological state that damages afferent or efferent nerve paths from thermo-receptors to target organs, or affects the centre for thermal control in the hypothalamus, can partly or wholly eliminate the body's thermal defences. The resulting hypothermia or hyperthermia is health threatening, and in extreme cases life-threatening. Hypothermia, therefore, demands corrective treatment to restore a normal core temperature. This task can prove highly intricate.

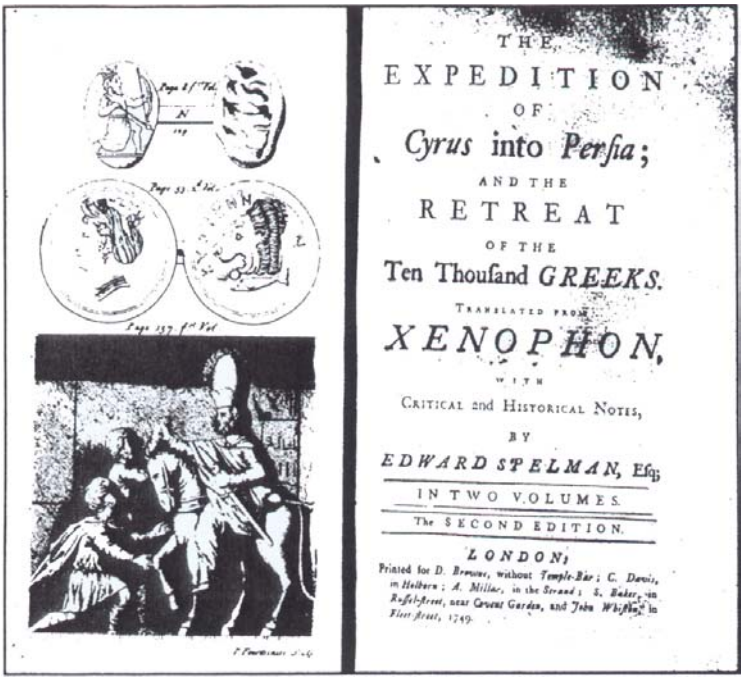
History

Scientific evaluation of cold and its effects on the human body began at the end of the 18th century. Before that, cold had been used for centuries in the treatment of various complaints, and especially fevers, inflammation and convulsions. Accidental hypothermia must have been common, even though it was not always recognised as such. Perhaps one of the earliest references to hypothermia appears in the Bible, in Chap. 1, bv. 1–4 of the First Book of Kings.

> *Now king David was old and stricken in years; and they covered him with clothes, but he got no heat.*
>
> *Wherefore his servants said unto him, Let there be sought for my lord the king a young virgin: and let her cherish him, and let her lie in thy bosom, that my lord the king may get heat.*
>
> *So they sought for a fair damsel throughout all the coasts of Israel, and found Abishag a Shunammite, and brought her to the king.*
>
> *And the damsel was very fair, and cherished the king, and ministered to him: but the king knew her not.*

Hypothermia figures in many other writings, for example those of Hippocrates, Aristotle and Galen, not always in an enlightened vein, and its treatment was always controversial (Paton 1983, Danzl 1988, Danzl et al 1988).

The best accounts of hypothermia are to be found in military history. In many battles, cold has had a decisive effect upon the result. In 401 B.C. Xenophon took part in the campaign of Cyrus the Younger and, together with 10,000 Greek soldiers, marched from Sardis to Babylon (Baghdad) and back through the Armenian Mountains. In one of the oldest historical documents recording the decimation of an army by cold, it was described how many of these soldiers suffered frostbite, resulting in amputation and death (Spelman 1749).

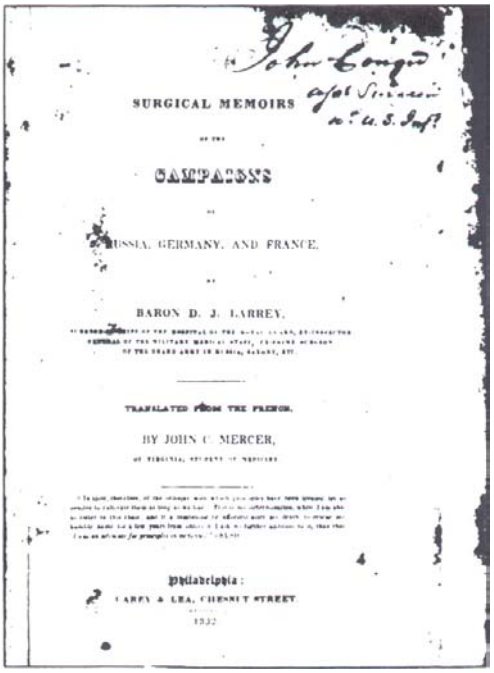


THE
EXPEDITION
OF
Cyrus into *Perſia*;
AND THE
RETREAT
OF THE
Ten Thouſand *GREEKS*.
TRANSLATED FROM
XENOPHON,
WITH
CRITICAL and HISTORICAL NOTES,
BY
EDWARD SPELMAN, Eſq;
IN TWO VOLUMES.
The SECOND EDITION.
LONDON:
Printed for D. Browne, without Temple-Bar; C. Davis, in Holborn; A. Millar, in the Strand; S. Baker, in Ruſſel-ſtreet, near Covent Garden, and John Whiſton, in Fleet-ſtreet, 1749.

Fig 2. One of the oldest historical documents recording the decimation of an army by cold (Spelman 1749).

Alexander the Great, so rumour had it, went into a coma caused by hypothermia on one of his campaigns, as did many Roman legionnaires when crossing the alpine passes. Hannibal lost 20,000 of his 46,000 men in 218 B.C. when they crossed the Pyrenees in Northern Spain and then traversed the Alps, descending into the Po Valley (Bangs and Hamlet 1983). Napoleon started out in 1812 with 650,000 troops, but by the time he left Moscow in October 1812 only 110,000 remained, and of these barely 2000 reached France fit enough to fight. Thousands died as a result of cold and wounds. During this campaign his surgeon general, Baron D.J. Larrey, introduced rubbing with snow as therapy for frostbite, a treatment which, although mistaken, continued to be used by the Swedish army well into the present century. The 1932 Swedish army medical handbook states: "Bathing in ice-cold water is recommended for general hypothermia." For local frostbite: "Rub with snow until warmth and feeling return." An army order from 1958 under the heading "Rules to be remembered by medical orderlies" reads: "Thaw out with massage, but not with snow." Larrey observed that "those placed nearest the camp fire mysteriously died," which is an important observation, knowing what we do today about "rewarming shock".

Many of Napoleon's soldiers survived by slaughtering a horse and sheltering in the warmth of the carcass, but many were trapped in this way because the carcasses froze during the night and they were unable to extricate themselves (Larrey 1832; Brantigan and Paton 1978).


SURGICAL MEMOIRS

CAMPAIGNS

RUSSIA, GERMANY, AND FRANCE.

BARON D. J. LARREY,

TRANSLATED FROM THE FRENCH.

BY JOHN C. MERCER,

Philadelphia:

CAREY & LEA, CHESNUT STREET.

1832

Fig 3. A detailed and highly empirical description of the effect of cold on a retreating army (Larrey 1832).

More instances of hypothermia are described in Swedish history. In August 1718, Carl Gustaf Armfelt, with 10,750 men under his command, marched from Duved in the Swedish province of Jämtland on Trondheim in Norway, which however, he failed to capture. During his retreat in the New Year 1719, his army, now decimated to 5,150, was caught in a blizzard and only about 1,700 men survived the crossing of the mountain wastes. In 1756, Samuel Naucler, a district medical officer, published an excellent description of the clinical picture of hypothermia, recording the treatment of "one man who, although apparently frozen to death, was restored to life" (Naucler 1756) (See App. 5).

During the Crimean War (1854–56), more than 1,000 French soldiers were lost to hypothermia. In the First World War, 115,000 British and 80,000 French troops suffered frostbite or trench foot. In the Second World War, no fewer than 90,000 American and more than 200,000 German troops were disabled by various cold-related injuries.

In spite of these experiences and the use of modern clothing and modern equipment, cold continues to cause injuries in modern warfare. During Israel's Yom Kippur War in 1973, large numbers of soldiers suffered frostbite on the Golan Heights. At Goose Green, during the Falklands War of 1982, when the temperature fell at most to -4 °C at night, hypothermia, frostbite and, above all, non-freezing cold injuries (NFCI) were common (Francis 1984; Andrews 1987). Talking about the Gulf War of 1991, Rear Admiral William Mills of Anchorage stated that heavy losses could very well have been sustained from the cold if Baghdad had been

taken and the allies had been forced to move up into the Iraqi mountains, because the troops had summertime uniforms only (personal information). The problems of cold were later to affect the Kurds in the Iraqi mountain ranges.

Epidemiology

Accidental hypothermia is defined as unplanned reduction of body core temperature to below 35 °C (Mackowiak et al 1992). When talking about hypothermia it is practical to divide cases into primary (healthy persons who are unexpectedly exposed to cold) and secondary (persons who have been injured or have contracted a disease, e.g. cancer, sepsis, hypothyroidism, which predisposes them to or causes, hypothermia).

In Sweden 30–45 hypothermia fatalities occur per annum (Albiin and Eriksson 1984; Statistics Sweden 2000). The number may actually be greater, because a number of persons classified as "drowned" while wearing a life jacket may have died of hypothermia. Similarly, a number of elderly persons found dead outdoors and not autopsied but allotted a presumed diagnosis of cerebrovascular or cardiovascular disease, may have died of hypothermia.

Fatal hypothermia in the civilian population mainly affects three groups. The first and largest of these groups consists of persons under the influence of alcohol (Hirvonen and Huttunen 1976; Albiin and Eriksson 1984). The second group, persons with pshyiatric disorders or dementia has not been earlier clearly recognised The third group is composed of outdoor enthusiasts meeting with accidents in potentially dangerous conditions: people immersed in cold water, stranded or lost in cold and windy weather, or injured, so they cannot find their way to shelter.

Hypothermia involving mountain hikers is not uncommon in Sweden. One such event was the Anaris disaster of 1978, in which eight young people lost their lives. More recently, in 1998 a family was stranded near Treriksröset in the middle of a winter storm. The father went for help while his wife and children remained in their tent. The tent became buried and the rescuers only found the family because of a hand protruding above the snow. All survived but the wife and children had profound hypothermia and the father suffered severe frostbite with loss of toes.

The accident on Mt Hood in Oregon, USA, in 1986 is one more tragic instance of the dangers of cold and unexpectedly severe weather. On that occasion eleven young people were trapped in a snow cavern when they were caught by a severe snowstorm while trying to climb Mt Hood (Wilkerson and Hamlet 1988). Nine died and two survived.

The events on Mt Everest in May 1996,in which nine climbers from four different expeditions lost their lives, are an extreme example of hypothermia and frostbite causing death in the mountains. Exhaustion, extreme cold, hypoxia, inexperience and poor judgement all played their parts in the fatal outcome (Krakauer 1998).

The overall incidence of hypothermia in the Netherlands is 1.1 per 100,000 of the population, which is similar to the incidence in Sweden. The Dutch have identified two groups of victims; those who become hypothermic because of immersion in cold water and those who become cold under other circumstances. The cold water group is younger (38.9 years vs. 55.2 years) than the non-water group. Trauma was associated with both groups. The immersion patients died as a result of submersion, the non-submersion patients died as a result of complications of hypothermia (Bierens 1995).

Hypothermia in connection with accidents in sparsely populated areas, especially during the cold season of the year, is doubtless more common than we know. Severe injuries adversely affect, or entirely eliminate, thermoregulation, resulting in an increase in mortality (Jurkovich 1987). The chain of reflexes from the dermal receptors to the temperature control centre in the hypothalamus can be broken, the feedback system interrupted and the defence of the individual against hypothermia compromised. When this happens, the individual passively loses heat to his surroundings and his body temperature quickly declines accordingly.

Predisposing factors

Several factors contribute to the development of hypothermia. These can be roughly divided into those reducing heat production, those increasing heat loss and those impairing thermo-regulation.

Conditions reducing heat production

Old age is accompanied by reduced muscular mass and impaired neuromuscular co-ordination, both of which reduce the capacity for shivering (Young 1991). Elderly persons are also less able to increase their metabolic heat production and respiratory quotient (Goldman et al 1977; Maclean and Emslie-Smith 1977; Reuler 1978). *Newborns*, within the first few hours of life, have difficulty in increasing heat production in response to heat loss. Their large surface area in relation to body mass and a sparse subcutaneous layer of fat contribute to their vulnerability. Within five days, however, the newborn has adjusted completely to its surroundings and, if exposed to cold, can rapidly augment its lipolysis and quickly convert its store of brown fat into heat (Perlstein et al 1974; Himms-Hagen 1984; Robinson and Seward 1986; Iyengar and Bhakoo 1991).

In certain *endocrine diseases*, such as hypothyroidism and pituitary insufficiency, both heat production and energy consumption are reduced. The net result, however, is impaired capacity for maintaining body temperature in cold conditions. *Starvation states* of various kinds reduce the subcutaneous fat, body insulation deteriorates, and the capacity for compensatory combustion is limited.

States of increased heat loss

An intact skin is protection against our surroundings. *Skin disorders* like psoriasis and dermatitis increase heat losses (Grice and Bettley 1957; Krook 1960; Reuler et al 1977). Burns have the same effect, both by damaging the skin and causing increased evaporation from the burned surface. Customary treatment with cold water or damp cloths accentuates heat loss (Livingston and Groggins 1984).

Alcohol can increase the risk of hypothermia . Although alcohol confers a feeling of warmth and well-being, one study from the north of Sweden found alcohol to be the commonest contributor to hypothermia. In that study, roughly two-thirds of all fatal hypothermia victims were under the influence of alcohol (Albiin and Eriksson 1984). Alcohol inter-

acts with every known neuro-transmitter, including serotonin and dopamine. Alcohol dilates the vessels of the skin to a certain extent and, in this way, augments heat losses, however, when exposed to a significant cold stress, the inhibitory effects of alcohol are overridden unless intoxication is severe (Johnston et al 1996). Alcohol augments the risk of exposure to cold by impairing sense of judgement and by clouding consciousness. Alcohol reduces the freezing point of cells, and may reduce the risk of local frostbite. Persons under the influence of alcohol have been known to survive at lower body temperatures than sober victims, due possibly to a sympatholytic effect with less risk of ventricular fibrillation (Huttunen and Hirvonen 1977; Granberg 1991).

States of impaired thermo-regulation

Injuries to the central nervous system (CNS) can directly affect the functioning of the hypothalamus and mediate vasodilation, thus leading to heat loss. Traumatic injuries such as intracerebral haemorrhage and subdural haematoma and pathological states such as Parkinson's disease, Alzheimer's disease and neoplasms, can have an adverse influence on thermo-regulation (Fox et al 1970; Chang and Gill 1981). Anorexia nervosa patients suffer from inadequate thermo-regulation are poorly nourished and have a sparse layer of subcutaneous fat and so they are more prone to hypothermia (Mecklenburg 1974).

Cross-sectional lesions of the spinal cord eliminate peripheral thermo-regulation distal to the injury. The injured person becomes functionally poikilothermic within the affected area when the capacity for vascular constriction is eliminated (Pledger 1963; Ashworth et al 1982; Altus et al 1985; Menard and Hahn 1991).

Neuropathies, as in diabetes mellitus, impair thermo-regulation. The elevated plasma osmolality in uncontrolled or poorly controlled diabetes mellitus, appears to have a central effect on the hypothalamus. This is especially noticeable in hypoglycaemia and ketoacidosis, and also in uraemic states (Swartz et al 1983; Neil et al 1986; Guerin and Meyer 1987; Jonsson and Gamble 1991).

Several medications, in both therapeutic and toxic doses, have a negative effect on central thermo-regulation. The most common drugs include benzodiazepines, phenothiazines and cyclic antidepressants (Kallenback et al 1981). But even drugs such as erythromycin, clonidine, flufenazine, atropine and beta blockers can have an adverse effect on peripheral thermo-regulation. An overdose of any drug or medication that induces coma will affect thermo-regulation leading to the possibility of hypothermia. *Severe trauma* has a profound effect on thermo-regulation. Injuries to the skin and muscles might impair or eliminate signals

from the peripheral thermoreceptors. Hypovolaemia and hypotension have an effect on central thermo-regulation. Wet or blood-soaked clothing augments heat loss. In one study by Jurkovich et al (1987) in which injuries were classified on an Injury Severity Score (ISS), an anatomical injury classification, hypothermic patients were shown to have higher mortality than similarly injured normothermic subjects. In a similar study, but this time employing TRISS methodology (a combination of physiological parameters and ISS), the finding could not be verified (Steinemann et al 1990). Possibly TRISS is a less reliable scale for hypothermia. For instance, the TRISS scale might interpret low blood pressure as being a sign of severe injury; while in fact the low blood pressure could be due to hypothermia and so the scale might overestimate the severity of the injury. Hypotension, for example, is normal at lower body temperatures (Danzl et al 1995).

Not only is there a great risk of hypothermia in association with traumatic injuries, but hypothermia also affects the traumatic injury. Hypothermia can cause a severe coagulopathies. The coagulation cascade is adversely affected, the thrombocyte count declines and their function is impaired, until finally a disseminated intravascular coagulation DIC syndrome ensues (Kashuk et al 1982; Sabapathi et al 1986; Britt et all 1991; Ferraro et al 1992; Rohrer and Natale 1992).

Heat loss and ambient factors

Physiological heat losses

The human body produces heat, approximately 80–90 W at rest, equalling the power of a filament bulb. Moderate physical exertion can triple this figure and heavy physical exertion can raise it ten times. Most of this heat is dissipated through the skin. Heat loss from the surface of the body takes place through convection (movement of wind or water), conduction (contact with cold objects or liquids), radiation, which is minimal with good clothing insulation, and the evaporation of sweat or water .

At least 580 kcal (2,430 kJ) are lost per litre of evaporated perspiration. This is an effective restraint on temperature increase during heavy physical exertion, when perspiration can amount to one or two litres per hour. Insensible perspiration, 750–1,200 ml/day, from the lungs and upper airway is another source of evaporative heat loss.

During exposure to cold, blood flow to the skin and therefore heat loss are reduced by vasoconstriction. This primarily affects the hands and feet. With vasoconstriction at its most pronounced, perfusion of the skin is reduced from the basal blood flow of 200–500 ml/min to as low as 20–50 ml/min.. The blood vessels to the head are sparingly supplied with sympathetic fibres, and these disappear entirely when the vessels enter the parenchyma of the brain – a practical arrangement because otherwise, in a cold climate, the supply of blood and heat to the brain would be reduced. On the other hand, this causes heavy heat losses due to the copious perfusion of blood through the head. In an otherwise fully dressed person at rest, but bareheaded, 50 per cent of heat loss occurs through the head at -10 °C and even more at -20 °C (Froese and Burton 1957).

In other words, the body has a big “hole in the head” through which heat can escape. Heat is also lost through the respiratory tract but mostly from evaporation and not by cooling the mucus membranes (Goheen S et al 1997). Most of the cooling by wind and water affects the torso. Heat loss from the torso increases greatly if the insulative value of clothing is deminished by wind and water.

Loss of heat in water

The thermal conductivity of water is up to 25 times greater than air of the same temperature. This leads to high rates of heat loss even in relatively warm water. In water at a temperature below 30–32 °C, thermo-neutrality is threatened and metabolism increases. The speed with which critical

water temperature is reached, i.e. the point at which the core of the body begins to grow colder, depends on several factors including the thermal insulation of clothes, the thickness of subcutaneous fat, the ratio of body surface area to mass, the temperature and conditions of the water (Smith and Hanna 1975, Steinman 1995). The highest critical water temperature noted by these authors in a thin subject was 35 °C. In very obese people, the critical water temperature may be less than 30 °C.

Below critical water temperature, metabolism and heat production increase linearly in relation to the reduction of water temperature. This, however, is an uneven struggle. When body temperature falls below 32 °C, the shivering mechanism is blunted and even eliminated. Deliberate efforts to increase heat production by movement, such as swimming or treading water, lead to increased heat loss by convection. The victim is in a vicious circle, leading to further reduction in core temperature.

Several authors (Molnar 1946; Keatinge 1969; Hayward 1975(a); Boutelier 1979; Tikuisis 1997) have attempted to predict survival time in water at different temperatures. However, the dependability of these times can be greatly reduced by waves, currents and traumatic injuries.

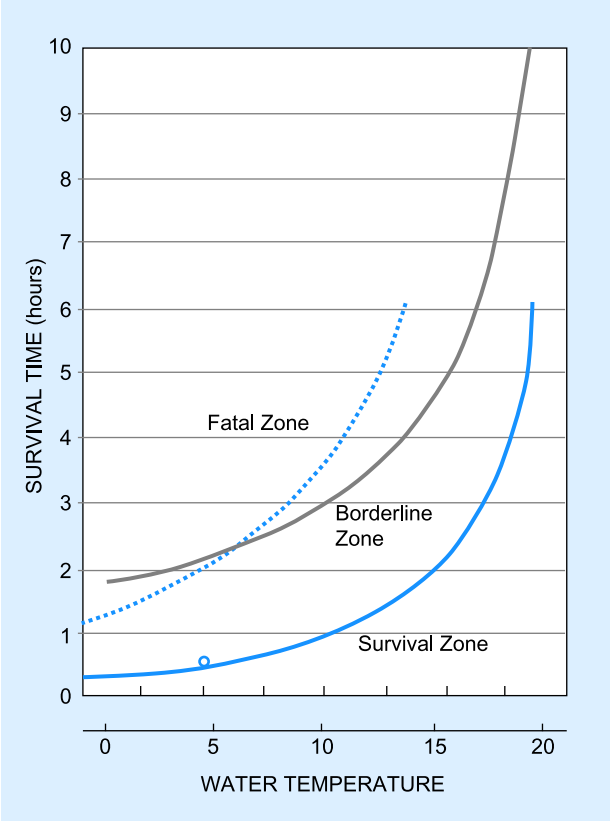


Fig 4. Survival in water as a function of temperature and time. The "Borderline Zone" indicates water temperatures at which 50% can be expected to lose consciousness, and which more often than not leads to drowning. "Fatal Zone" indicates the water temperatures at which no survival is expected. The continuous line predicts 50 % survival, based solely on hypothermia (cart modified after Molnar 1946; Hayward 1975, 1984).

Experience of the sinking of the M.V. Estonia on 28th September 1994, when 859 people died, and only 137 survived, suggests that Hayward's and Keatinge's figures are perhaps an underestimate and that there are unpredictable differences between survivors. Survival times for certain strong-willed individuals are probably longer than was predicted. Unconsciousness (due to hypothermia) and turbulent waves lead to a great risk of drowning by aspiration, when the unconscious victim is rolled about in the water. Heat loss is greatly increased by swimming or treading water, especially by swimming on the back with one's head in the water, due to the inability of the head to reduce heat emission by vasoconstriction. Several authors maintain that when water temperature is sufficiently low, hypothermia is more rapid during movement than at rest (Hayward 1975(b); Keatinge 1961; Hayward and Keatinge 1981). Swimming in 10.5 °C water caused heat production to rise 2.5 times, although hypothermia occurred despite this (Hayward 1975(b)). Studies of English cross-Channel swimmers in the 1950s showed rectal temperatures of 34–38.3 °C after 18 hours in water at 16 °C. This was attributed to the swimmers' fairly thick subcutaneous fat layers and to high metabolism while swimming (Pugh and Edholm 1960).

The buoyancy of water in a flowing stream or waterfall is diminished because of air bubbles, eddies and undertow. This means that in rough water even people with life-jackets can be pulled under the water and drowned quickly. In addition, sudden immersion in cold water may induce a rapid rise in respiratory rate, with a consequent change in blood pH, and increase the risk of ventricular fibrillation. In some people this reflex can be partly eliminated by training through repeated exposure to cold water.

Water temperatures below 21°C can seriously threaten temperature control. A person who has apparently drowned while wearing a life-jacket may be suffering from severe hypothermia and thus not be beyond saving. In cases where the “drowned” victim has been under the surface of water colder than 21ºC for less than 60 minutes, hypothermia may have protected the brain and the patient might be potentially resuscitable (Siebke 1975). This is truer with small children than with adults, but there is at least one report of an adult who was totally submerged for 15 minutes and recovered with only minor neurologicalal problems (Chochinov et al 1998). There have been several reports of children submerged for prolonged periods, up to 66 minutes, with total recovery (Sekar 1980; Bolte 1998). [see Diving Reflex, page 105]

In many instances when a person is immersed in cold water they are unable to swim for more than a few minutes before drowning. The core temperatures of these victims are not usually hypothermic, but muscle fatigue sets in rapidly and they can no longer swim. When such people are treated for near drowning, more attention should be paid to the prob-

lems of hypoxia and drowning than to their mild hypothermia (Tipton et al 1999).

Sudden immersion in cold water initiates an inspiratory gasp response, followed by uncontrollable hyperventilation and tachycardia. This "cold shock" response can be dangerous in conjunction with a dive or jump into very cold water, as the respiratory drive could induce breathing before the surface is reached.

Wind effects

Wind increases the risk of hypothermia and frostbite. The hypothermic effect is due to convection, whereby the wind, as its velocity increases, removes warmed air surrounding the exposed subject with increasing speed. Even low wind velocities accelerate the rate of heat loss, due to the removal of the insulating layer of heated air from round the body. This causes heat loss, so the victim is compelled to supply additional heat to the skin to keep its temperature from being reduced to ambient temperature. If severe, such conditions may cause local cold injuries to exposed parts of the body, such as the face. Prolonged exposure can lead to a general loss of body temperature. Dr Paul Siple (1945) calculated the combined effect of wind and cold in a "wind chill index". This index shows the temperature required in calm weather to produce the same cooling effect as a certain wind velocity and temperature combined.

Table 1. Table showing the hypothermic effects on bare skin allowing for wind strength. Example: -10 °C combined with a wind strength of 15 m/sec corresponds to -33 °C in calm weather (after Paul Siple).

Wind velocity (m/sec)	Temperature °C						
	0	-5	-10	-15	-20	-25	-30
	Corresponding temp. loss effect on unprotected skin in calm weather						
calm	0	-5	-10	-15	-20	-25	**-30**
slight wind (1.5–3.5)	-4	-14	-20	-23	-26	**-28**	**-33**
moderate wind (3.5–8)	-10	-21	-25	**-32**	**-38**	**-45**	**-52**
fresh wind (8–14)	-15	-25	-28	**-36**	**-48**	**-56**	**-63**
strong wind (14–20)	-18	-27	**-33**	**-38**	**-51**	**-57**	**-65**
half gale (21–25)	-19	-28	**-36**	**-43**	**-52**	**-60**	**-68**
Risk:	Negligible risk of frostbite	Increasing danger	Great danger: rapid hypothermia, from a few minutes down to a few seconds				

Wind-chill has been re-calculated, based on new experiments. Although the numbers have changed slightly, the principle of wind cooling remains the same (Osczevski 1995, Danielsson 1996).

During 2001, a team of scientists and medical experts from Canada and the U.S. worked together to develop a new wind chill index. The research agency of the Canadian Department of National Defence, with its knowledge of how troops are affected by cold weather, contributed to the effort by conducting experiments using human volunteers.

The new index is based on the loss of heat from the face-the part of the body that is most exposed to severe winter weather. Volunteers were exposed to a variety of temperatures and wind speeds inside a refrigerated wind tunnel. They were dressed in winter clothing, with only their faces exposed directly to the cold. To simulate other factors affecting heat loss, they also walked on treadmills and were tested with both dry and wet faces.

To ensure the new wind chill index meets the needs of Canadians, Environment Canada conducted public surveys across the country. The new index is expressed in temperature-like units, the format preferred by most Canadians.

However, since the wind chill index represents the feeling of cold on the skin, it is not actually a real temperature, so it is given without the degree sign. For example, "Today the temperature is -10°C, and the wind chill is -20".

The following tables are taken from The Meteorological Service of Canada and represents the most up to date calculations for wind chill. This table which has been accepted by authorities in Canada and the United States should be used for calculating wind chill in the place of older tables.

Wind Chill Calculation Chart

/T air	5	0	-5	-10	-15	-20	-25	-30	-35	-40	-45	-50
V_{10} km/h												
5	4	-2	-7	-13	-19	-24	-30	-36	-41	-47	-53	-58
10	3	-3	-9	-15	-21	-27	-33	-39	-45	-51	-57	**-63**
15	2	-4	-11	-17	-23	-29	-35	-41	-48	-54	**-60**	**-66**
20	1	-5	-12	-18	-24	-31	-37	-43	-49	-56	**-62**	**-68**
25	1	-6	-12	-19	-25	-32	-38	-45	-51	-57	**-64**	**-70**
30	0	-7	-13	-20	-26	-33	-39	-46	-52	-59	**-65**	**-72**
35	0	-7	-14	-20	-27	-33	-40	-47	-53	**-60**	**-66**	**-73**
40	-1	-7	-14	-21	-27	-34	-41	-48	-54	**-61**	**-68**	**-74**
45	-1	-8	-15	-21	-28	-35	-42	-48	-55	**-62**	**-69**	**-75**
50	-1	-8	-15	-22	-29	-35	-42	-49	-56	**-63**	**-70**	**-76**
55	-2	-9	-15	-22	-29	-36	-43	-50	-57	**-63**	**-70**	**-77**
60	-2	-9	-16	-23	-30	-37	-43	-50	-57	**-64**	**-71**	**-78**
65	-2	-9	-16	-23	-30	-37	-44	-51	-58	**-65**	**-72**	**-79**
70	-2	-9	-16	-23	-30	-37	-44	-51	-59	**-66**	**-73**	**-80**
75	-3	-10	-17	-24	-31	-38	-45	-52	-59	**-66**	**-73**	**-80**
80	-3	-10	-17	-24	-31	-38	-45	-52	**-60**	**-67**	**-74**	**-81**

R. Osczevski 1995, Defence R&D Canada

where T_{air} = Actual Air Temperature in °C
V_{10} = Wind Speed at 10 metres in km/h (as reported in weather observations)

Thresholds:

Risk of frostbite in prolonged exposure	-25	
Frostbite possible in 10 minutes	-35	Warm skin, suddenly exposed. Shorter time if skin is cool at the start.
Frostbite possible in 2 minutes	**-60**	Warm skin, suddenly exposed. Shorter time if skin is cool at the start.

Minutes to frostbite for the 5% most susceptible segment of the population

Temperature (˚C)	-15	-20	-25	-30	-35	-40	-45	-50
Wind (km/h)								
10	*	*	22	15	11	8	7	6
20	*	*	14	10	7	6	5	4
30	*	18	11	8	6	4	4	3
40	42	14	9	6	5	4	3	2
50	27	12	8	5	4	3	2	2
60	22	10	7	5	3	3	2	2
70	18	9	6	4	3	2	2	2
80	16	8	5	4	3	2	2	1

R. Osczevski 1995, Defence R&D Canada

* = Frostbite unlikely

The wind speed, in km/h, is at the standard anemometer height of 10 metres (as reported in weather observations).

Legend:

Frostbite possible in 2 minutes or less	2
Frostbite possible in 3 to 5 minutes	5
Frostbite possible in 6 to 10 minutes	10

The effect of wind chill is seen in connection both with “natural wind” and others such as a slipstream, the turbulence caused by aircraft propellers and the downwash from helicopter rotors. Standing under the slipstream of a helicopter in very cold weather can rapidly cause frostbite. Accident victims being carried into a helicopter must be adequately protected from the slipstream. Wetness combined with wind further accelerates heat loss from the body.

Conduction

Direct conduction of heat to surroundings or cold objects can cause significant loss of heat. This is especially important when traumatised patients are left lying on cold ground without adequate insulation beneath them.

Pathophysiology

The nervous system

The neurones of the central nervous system (CNS) are initially stimulated by exposure to cold, but subsequently the metabolism of the brain undergoes a linear decline of 6–10 % per °C at a body temperature between 35 and 25 °C (Michenfelder and Milde 1991). EEG becomes abnormal below 33 °C and shows no signs of activity at temperatures below 19–20 °C (Ehrmantraut et al 1957; FitzGibbon et al 1984). Under these circumstances lack of electrical activity does not imply brain death, merely suspension of activity. Cerebrovascular auto-regulation remains intact until the core temperature falls below 25 °C.

The desire to find warmth, shelter and food is perhaps the first clinical manifestation of declining body temperature. Memory and capacity for judgement deteriorate at an early stage of cooling, wrong conclusions are drawn, speech becomes slurred and consciousness becomes clouded. Confusion and quasi-psychotic, hallucinatory states occur at lower core temperatures. Consciousness declines and most subjects are comatose at 30 °C. But this boundary is not absolute and can vary in both directions. Patients with a rectal temperature as low as 28 °C may still reply when spoken to. Hyperreflexia is seen at 35–32 °C, after which hyporeflexia ensues. The pupils dilate at 32 °C, and eye movements, light reflexes, and tendon reflexes disappear at 27 °C.

The circulatory system

The heart

Cold initially causes tachycardia and peripheral vascular constriction. This is followed by increasing bradycardia and at 28 °C bradycardia with a heart rate about half normal may occur (Blair 1964; Sinet et al 1985). This bradycardia is caused by decelerated spontaneous depolarisation of the pacemaker cells, and accordingly is not amenable to atropine treatment (Preston 1976). Cardiac output and mean arterial blood pressure gradually decline until the heart stops in asystole or ventricular fibrillation. If a viable rhythm still persists at 25 °C, cardiac output is 45 per cent of normal at most. Even after rewarming, cardiovascular function, including contractility, metabolism and peripheral vascular function may not immediately return to normal.

EKG

Initially the PR interval is prolonged, followed by widening of the QRS and, most characteristically, the QT interval. A J-wave (Osborn wave), most often a positive deflection in the QRS-ST transition, may occur when the temperature falls below 32 °C. It occurs most frequently in leads II and V6. The size of the J-wave increases with falling temperature. This can be diagnostic, but is not prognostic. The J-wave is sometimes seen in CNS injuries and in patients with sepsis. Thus it is not pathognomonic for hypothermia. When pronounced the J-wave can simulate cardiac infarction, an important distinction to make, given the increasing use of thrombolysis in pre-hospital care (Glusman et al 1990). Emergency thrombolytic therapy would be highly undesirable in a hypothermic patient with a compromised coagulation system. Dr Mills of Anchorage, who has had much practical experience of treating hypothermia cases, maintains however, that in practice the J-wave is not seen as often as is described in the literature.

Arrhythmias

Arrhythmias of all kinds can be expected below 32 °C during both cooling and rewarming (Emslie-Smith 1958; Duguid et al 1961; Edwards et al 1970; Clements and Hurst 1972). Atrial arrhythmias are presumed to be triggered by atrial distension, whereas ventricular arrhythmias probably have a multifactorial genesis. The His-Purkinje system is more sensitive to hypothermia than the myocardium, which leads to decelerated conduction and, potentially, to the diffusion of electric signals. The deceleration of conduction, beyond the absolute refractory period, can contribute to a re-entry phenomenon, which in turn can result in both ventricular ectopy and ventricular fibrillation. Hypothermia increases both the height and the duration of the action potential (Björnstad 1991). This, coupled with decelerated conduction time in an unevenly cooled myocardium, can lead to blockage, re-entry phenomena and arrhythmias. Finally, isolated electrical foci in the hypothermic heart can trigger arrhythmias (Covino and Beavers 1958). Electrolytic disturbances, especially hyperkalaemia, can further complicate the situation.

A hypothermically induced "calcium overload" in the myocardial cells has also been measured. This can predispose to a "Torsades de pointes"-like arrhythmia. Hypothermically induced asystole and atrial fibrillation are probably due in part to re-entry phenomena, calcium overload and isolated electrical foci. Both asystole and ventricular fibrillation can occur spontaneously below 25 °C. Certain authors maintain that asystole is the most common, while ventricular fibrillation is iatrogenic (Southwick and Dalglish 1980; Bangs 1984; Ferguson 1985). The end result is the same

regardless of the final rhythm. Both states have as potential etiologies, hypovolaemia, hypoxia, therapeutic interference: e.g. precordial stimulation during handling, endotracheal intubation central catheterisation, acid-base disturbances, autonomous dysfunction and coronary vasoconstriction (Swan et al 1953; Vandam and Brunap 1959; Westin et al 1961; Mouritzen and Anderson 1965; Lloyd and Mitchell 1974; Swain et al 1984).

Blood vessels

Peripheral vasoconstriction is the body's initial defence against heat loss. Cold is first felt in the hands, feet and face.With cold stress, arteriovenous shunts in the hands and feet are closed, thus greatly decreasing the flow of blood, reducing heat loss from the skin and preserving core temperature and circulation. Blood flow to half the body's surface area and a third of its volume is thereby greatly reduced. Peripheral circulation might be reopened for a limited period, permitting warm blood to flow through the extremities for a short time. In this way the body might preserve some use of the hands and feet for a time, while still saving heat. Given good thermal balance, this can be repeated for 3–4 cycles ,"Lewis Hunting Wave phenomenon", (Lewis 1930; Vangaard 1990). With falling body temperature, this fades and a persistent peripheral vasoconstriction ensues. Peripheral vasoconstriction leads to an increase in the volume of blood in the capacitance vessels of the core of the body, which in turn leads to increased "cold diuresis" and to increased leakage of fluid into the interstitial space. In some victims a vasodilatation occurs, most often preterminally, that may result in a feeling of extreme warmth, causing them to remove their clothes (Wedin, 1979).

After-drop

The term "after-drop" denotes a continuing fall in temperature after the hypothermic patient has been removed from the cold environment.Two factors contribute to this phenomenon.The first is continuing equalisation between the warm core of the body and the periphery. The other is the return of cold blood to the core reducing central temperature (Davies et al 1967; Webb 1973; Hayward and Steinman 1975; Golden and Hervey 1977; Reuler 1978; Jessen 1979; Harnett et al 1980; Harnett et al 1983; Steinman 1987). After-drop is, thus, a combination of two processes, a partial transfer of heat between cold and warm areas, and the transfer of heat through circulation (Giesbrecht and Bristow,1992). After-drop is seen at its most pronounced when there is a big difference in temperature between the periphery and body core and when blood flow to the periphery increases. Steep after-drop has been observed during the thawing of frozen extremities, prior to the stabilisation of core temperature.

The importance of after-drop depends on the lowest temperature reached and the magnitude of the fall in temperature. An after-drop of 1.5 °C from 34 °C to 32.5 °C is of little clinical danger. But a similar after-drop from 30 °C to 28.5 °C would take the patient into a temperature zone where cardiac arrhythmia would be very likely.

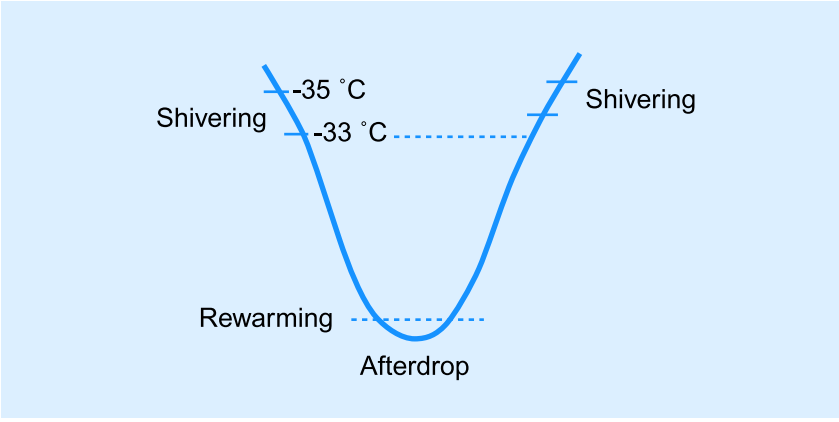


Fig 5. After-drop is the part of the curve indicating a persistent fall in temperature despite the application of warming. Attention should be paid to the slight shift in the position of shivering connected with loss of temperature and rewarming, respectively. The point at which shivering begins during rewarming is determined not only by body temperature but also by glucose availability. If shivering begins but later stops, glucose and/or insulin deficiency must be suspected and remedied (Hamlet, personal communication).

The Coagulation System

Hypothermic patients often develop coagulation disturbances and diffuse bleeding (Diaz et al 1984). One explanation is that the enzymatic systems of the coagulation mechanism are inhibited by the cold (Ferrara et al 1990; Reed et al 1990; Reed et al 1992). Hypothermia at temperatures below 33 °C produces a coagulopathy that is functionally equivalent to significant (<50% normal activity) factor-deficiency states under normothermic conditions, despite the presence of normal clotting factor levels (Johnston et al 1994).

Coagulation samples are analysed at 37 °C. This can lead to a disparate picture of normal values for coagulation specimens from a patient who is bleeding. The appropriate treatment may often be application of warmth, not coagulation factors.

Thrombocytopenia due to direct bone marrow depression and sequestration of platelets in the spleen and liver may also cause bleeding (O'Brien et al 1982; Pina-Cabral et al 1985; Rosenkrantz 1985). In induced hypothermia, the platelet count has been shown to decline on average from 184,000 to 37,000/ml (Shenaq et al 1986). Hypercoagulation occasionally occurs and conveys a similar picture to DIC. The mechanism is assumed to be thromboplastin release from hypothermic tissue, circulation collapse, release of catecholamines and steroids (Carden and Nowak 1982). When the skin is cooled there may be a transient increase in platelet function which could explain the heightened incidence of coronary and cerebral thromboses during winter (Keatinge et al 1984).

The Respiratory System

Exposure to cold, especially cold water, initially stimulates a gasp reflex followed by accelerated breathing. The respiratory rate can thus become high enough to cause a respiratory alkalosis leading to convulsions, semiconsciousness, ventricular fibrillation and a risk of drowning. As core temperature falls the respiratory rate and cardiac output diminish proportionately. Below 30 °C, the respiration rate is 5–10/min and breathing is shallow. If temperature declines further, respiratory regulation exercised by the medulla oblongata is affected. Carbon dioxide retention and respiratory acidosis are no longer capable of stimulating respiration. Ciliary mucus transport is reduced, leading to an increased chance of pneumonia. Pulmonary secretions become more viscous and "non-cardiogenic pulmonary oedema" may develop due to fluid leakage from pulmonary capillaries (Morales and Strollo 1993). This, de facto, can also be cardiogenic, in the sense of being triggered by a strong sympathetic discharge which leads to acute pulmonary hypertension and exudation of blood plasma into the alveoli, accompanied by pulmonary oedema. The thorax becomes less and less elastic, compliance diminishes and the respiratory muscles become less and less efficient. All these factors combined contribute to ventilatory insufficiency (Okada and Nishimura 1990).

Renal function and fluid balance

Exposure to cold entails changes in fluid balance that become more pronounced as body temperature declines. During early hypothermia, peripheral vasoconstriction, accompanied by an increase in central blood volume, leads to increased diuresis. This cold diuresis begins within 10 or 20 minutes of exposure to cold and can cause involuntary urination. Cold-diuresis has previously been interpreted as an aqueous-diuresis caused by reduced secretion of antidiuretic hormone. Human experiments have

shown, however, that cold-diuresis is an osmolar diuresis with sodium and chloride as its principal components (Lennquist 1972). The underlying mechanism of diuresis is the catecholamine secretion that is increased by exposure to cold (Lamke et al 1972). This leads to peripheral vasoconstriction, central blood volume increase and increased renal artery perfusion pressure. The peritubular capillar absorption capacity is a determinant factor in the transepithelial transport of sodium. A rise in capillary pressure , secondary to the increased renal artery perfusion pressure, increases the hydrostatic gradient against which sodium is transported, giving rise to reduced sodium reabsorption. Sodium is therefore lost in the urine, with accompanying fluid losses (Lennquist 1971, Granberg 1991). Cold-diuresis causes progressive haemoconcentration and reduced blood volume. In addition to urinary fluid loss, a pronounced extravasation of fluid into the interstitial space further reduces blood volume. The relative dominance of these mechanisms of volume redistribution varies according to the rapidity, duration and depth of hypothermia. Both mechanisms produce the same end result – hypovolaemia – which, in severe and prolonged hypothermia, can be severe. This has to be borne in mind when handling hypothermic patients. A hypothermic victim has often been fighting cold, snow and wind. Hard work in cold, dry air, increases loss of water, due to augmented ventilation and perspiration. It is often difficult to give replacement fluid in an outdoor environment. In addition, the victim frequently does not feel thirsty All these factors aggravate hypovolemia (Lennquist et al 1974). These factors have to be borne in mind when handling hypothermic patients. Careless handling in transit can lead to a loss of blood pressure sufficient to trigger arrhythmias and shock.

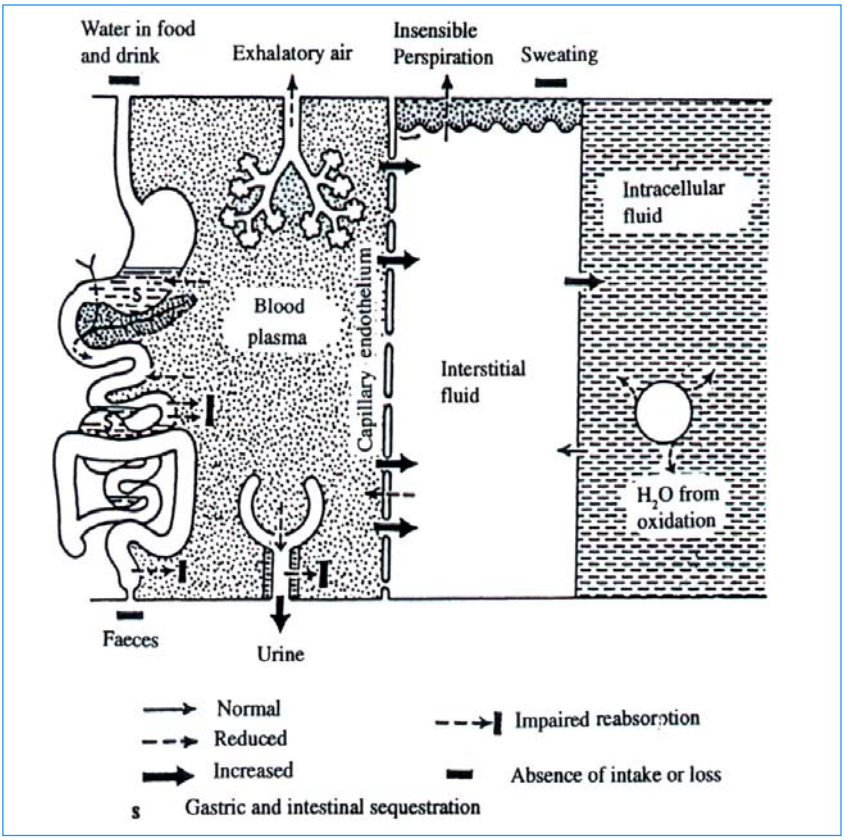


Fig 6. The effect of hypothermia on human fluid balance (modified illustration after Maclean and Emslie-Smith 1977).

Clinical picture

Hypothermia is easy to diagnose when somebody has fallen into icy water or been found in an avalanche. Diagnosis becomes more difficult when the hypothermia is mild and the exposure unclear. Early, vague symptoms include hunger, nausea, silence, fatigue, bad temper, lack of co-ordination and confusion. The degree of consciousness gradually declines, and even if rare, occasional people are still conscious and talking at 28 °C, especially children and persons under the influence of alcohol. The majority lose consciousness between 30–26 °C.

The skin turns cold and pale, especially at the extremities. The pulses, whether at the wrist, groin or in the neck becomes faint and slow and eventually becomes impalpable. Most often the blood pressure is impossible to measure, the heart sounds muffled, "far away" or inaudible. Respiration is slow and superficial. Hyperreflexia occurs between 35–32 °C, followed by hyporeflexia with areflexia occurring below 27 °C. The pupils also become fixed and dilated below 27 °C. Mental changes include early anxiety, poor judgement and neurotic behaviour. A patient rescued at this stage can have a disturbed perception of reality and subsequently may describe the situation in a way that differs from the true facts. Falling temperature brings apathy, hallucinations and psychosis (Sampson 1984).Preterminally, "paradoxical undressing" – removal of clothes without any natural explanation – is not uncommon (Wedin et al 1979, Albiin and Ericsson 1984) and may be due to receptor disturbance in the heat control centre of the hypothalamus. The finding of a naked victim is sometimes misinterpreted as the result of criminal action.

Muscular tone increases at the beginning of cooling, giving way to shivering which peaks at 35 °C. Shivering declines and disappears at about 33 °C. Shivering stops due to the exhaustion of the glycogen depots and direct hypothermia of the muscle cells. Other reasons for the absence of shivering could be the influence of drugs, alcohol or warming of the skin. Increasing rigidity follows, leading eventually to pronounced muscular stiffness which, at its most extreme, can resemble rigor mortis. In addition to cold and pallor, the skin is often frost-bitten, with excoriations and haemorrhagic oedema.

Table 2. Functional changes at different temperatures.

37 °C	Normal temperature
36 °C	Increased metabolism
35 °C	Maximum shivering. Hypothermia limit
33 °C	Shivering declines. EEG abnormal
32 °C	Cardiac arrhythmias, J-wave. Confusion
31 °C	Blood pressure barely noticeable
30 °C	Respiration declines, respiration rate 5–10/min. Semi-comatose
28 °C	Bradycardia, ventricular fibrillation can occur in response to stimuli
27 °C	Deliberate movement eliminated. Tendon reflexes disappear. No pupil reaction
26 °C	Most subjects unconscious. Respiration rate 4–7/min. Poikilothermia
25 °C	Spontaneous ventricular fibrillation or asystole can occur
20–19 °C	EEC reveals no activity

Laboratory findings

Blood status

Blood loss is easily misjudged, because haematocrit and haemoglobin levels rise as a result of cold-diuresis. The leucocyte count declines, owing to bone marrow depression and sequestration of white cells in the liver and spleen. Because of this the leucocyte count may be low or normal, even if the hypothermic patient becomes septic (Blair 1964; Lewin et al 1981; Shenaq et al 1986). The platelet count declines, due to direct bone marrow depression and sequestration of platelets in the spleen and liver (O'Brian et al 1982; Pina-Cabral 1985: Rosenkrantz 1985).

Acid base balance

In cases of acute hypothermia after sudden immersion in cold water, respiratory alkalosis, due to hyperventilation, may be so pronounced that convulsions ensue. This alkalosis is succeeded by an increasing respiratory and metabolic acidosis. The respiratory acidosis is due to respiratory depression. The metabolic acidosis is due to ketogenesis in the liver, lactate formation during shivering, impaired peripheral circulation, with accompanying tissue hypoxia, and a reduction in cardiac output. The proper interpretation of blood gas analyses has been a highly controversial topic. Should data be corrected to accord with the patient's current temperature or not? At one time Severinghaus' mathematical correction was used and the corrected value compared with the normal value of the patient's present temperature (Severinghaus 1966; Severinghaus and Astrup 1986). This is no longer applied. Instead the uncorrected blood gas values at 37 °C are simply compared with the normal values at 37 °C (Swain 1988; Swain et al 1990).

Optimum pH for blood at 37 °C is 7.42 and neutral pH for intracellular water at 37 °C is 6.8. Rahn proposed this pH difference of 0.6 should be retained at all temperatures. Because neutral pH rises during hypothermia, blood pH ought to do the same. Relative alkalosis in the tissues is physiologically appropriate and several studies corroborate Rahn's hypothesis, suggesting that an alkalosis retaining a pH difference of 0.6 between the interior of the cell and the blood should be aimed for at every temperature. If the pH difference is not maintained, the efficiency of the sodium-potassium pump deteriorates (Rahn 1974; Rahn et al 1975; Ream et al 1982; Baraka 1984; Baraka et al 1992). All in all, this implies that the cardiovascular and metabolic stability of the hypothermic patient is great-

est in a state of slight alkalosis. This state is most easily attained by analysis of blood gas values at 37 °C without correction to current temperature. Thereafter the patient's ventilation and acid base status are adjusted to pH 7.42 and PCO2 5.0–5.3, still at 37 °C. This leads to the desired alkalosis at the patient's present temperature, because blood pH rises with falling temperature. It has to be realised however, that the majority of acid base disturbances self-correct during rewarming, and surveillance and "wait-and-see" are therefore needed. Aggressive treatment of acidosis with bicarbonate should be avoided, because this often leads to excessive metabolic alkalosis during rewarming.

Electrolyte status

Serum electrolytes must be checked and continuously monitored during rewarming. Electrolyte values fluctuate with temperature, length of exposure and rewarming technique. There is no reliable trend. The potassium value is usually low in chronic hypothermia (Astrup et al 1981; Koht et al 1983; O'Connor 1986), but the opposite can occur. Hypokalaemia is mainly caused by potassium migrating into the muscle cells and only to a slight extent by kaliuresis (Boelhouwder et al 1987). Hyperkalaemia is occasionally seen in cases of acute hypothermia (immersion hypothermia). It is assumed to be caused by the sodium-potassium pump failing to function normally at temperatures below 30 °C, which leads to potassium leakage from the cells. Hyperkalaemia is more likely to be associated with crush injuries, renal failure, post-submersion haemolysis or pre-cooling death. Hypothermia and hyperkalaemia are potential risk factors for ventricular fibrillation, and serum potassium must therefore be continuously monitored. Prophylactic treatment with bicarbonate, insulin and/or calcium should be considered. For initial treatment however, intravenous fluid for dilution is recommended, with glucose and insulin as a second resort only. Prognostically, hyperkalaemia is a bad sign, suggesting pronounced cell damage. A serum potassium level of >10mmol/l, considered in association with other factors, is an indication of death and the inability to resuscitate the patient.

Hyponatraemia due to osmolar diuresis is common, especially in chronic hypothermia.

Blood glucose

Acute hypothermia initially raises blood glucose values through catecholamine-induced glycogenolysis. Prolonged exposure to cold, with shivering, fatigue and exhaustion of glycogen stores, later leads to hypoglycaemia. Cold-induced glycosuria is common and is not an indication of nor-

mo- or hyperglycaemia. In uncertain cases or where blood glucose measurement is not immediately possible, administer 30 ml 30 % glucose followed by 250–500 ml warm glucose 5 % (37–40 °C). Hyperglycaemia persisting during rewarming must prompt the suspicion of diabetic ketoacidosis or haemorrhagic pancreatitis. Insulin has no effect under 30 °C and therefore must not be given until the patient's temperature has been raised above that level. Otherwise there is a danger of iatrogenic hypoglycaemia developing after rewarming.

Other tests

Urea and creatinine values are often high, due to the inability of the hypothermic kidneys to secrete nitrogenous waste products. The amylase level is occasionally elevated (MacLean et al 1974) and may indicate associated pancreatitis. Ischaemic pancreatitis is attributed to microcirculatory collapse in the hypothermic pancreas. According to White (1982), the level of hyperamylasemia may correlate with potential mortality.

Treatment

When treating hypothermic patients consider whether hypothermia has developed rapidly or slowly, i.e. whether it is "acute" or "chronic". Treatment strategy will also depend on whether or not the patient is conscious.

In *acute hypothermia* for example, through immersion in cold water, temperature loss is so rapid there is no time for serious glycogen, fluid balance and electrolyte disturbances to develop. Rewarming, therefore, can proceed with less risk of hypovolaemia, fluid balance and electrolyte disturbances.

In *chronic hypothermia*, e.g. when a person gets lost in the mountains and suffers from a shortage of food and drink, major changes occur in the body. The glycogen stores are exhausted. Cold-diuresis and internal losses of fluid to the interstitial space lead to hypovolaemia. Acidosis and electrolyte disturbances develop. These changes have to be taken into account during rewarming, and appropriate action taken to monitor and correct the abnormalities.

Pre-hospital care

The first person to attend to a hypothermic patient may be confronted by difficult environmental conditions, such as a blizzard or a storm at sea. This can make it hard to perform even the first task, namely that of protecting the patient from further loss of heat and a fall in temperature (Mills 1992). The fundamental rules of action must be: *rescue, examine, dry and insulate, evacuate* (Steinman 1987).

It is unlikely that a doctor will be the first person to find the victim. In an urban setting the finder will probably be a neighbour or a paramedic. In the outdoors, the finder may be another hiker or skier. In a difficult outdoor situation it is important to remember that rescuers must not put themselves in danger. A second casualty does not help the injured person.

Since it can be very difficult, even for trained medical personnel, to decide on the best course of action, it is very important to establish early radio or telephone contact with a doctor who can supply instructions and suggest a course of action.

Conscious patient

The hypothermic but still conscious patient is often confused, with slurred speech, apathy and an abstracted look. All degrees of shivering can occur, from moderate to so severe that the patient is unable to lie still and

literally "shiver their teeth out". Sometimes, in patients equally cold, shivering may be absent in some and violent in others. Infant children, elderly persons and patients under the influence of drugs or alcohol do not usually shiver at all.

As far as possible, assessment of *the conscious hypothermic patient* must include a detailed history. When in an urban setting, ask questions about associated illnesses and injuries, drugs and medications, food and fluid intake. In the outdoors ask about injuries, duration of exposure and food and fluid intake over the previous few hours.

To ensure you address life-threatening conditions, assessment should follow steps A B C D E. To try and gather primary survey data in A B C D E order is unproductive. On the other hand, it is equally impossible to describe this logically in other than an orderly manner. The rest of this discussion on the assessment of the hypothermic patient will address the specific components that have been identified as adherent to the hypothermic condition.

Examination of the patient must include:

A – Airway management and cervical spine control
B – Breathing (ventilation)
C – Circulation
D – Disability
E – Exposure and environmental control, degrees and dry

The Airway should be checked for patency to ensure it is open and clear and that no danger of obstruction exists. Administer oxygen, 4–6 l/min on a bridle or 40–60 % with a mask, preferably warm and moisturised to reduce further heat loss and diminish the risk of arrhythmias (Budd 1986; Mills 1992; Bowman 1993; Lloyd 1996).

Cervical spine control – when checking and establishing an open airway, there is always the possibility that a cervical spine injury may exist. Thus early immobilisation is essential in most severely injured patients.

Breathing-Ventilation – Check spontaneity of ventilation. Respiration is often very slow, with a respiratory rate as low as 5–10 per minute. Tidal volumes can be very small and breathing superficial. When breathing is not spontaneous, administer ventilation.

Circulation – circulatory system failure is just as life-threatening as failure of the respiratory system. Evaluate the pulse for presence, quality and regularity. All pulses in the hypothermic may be extremely difficult to find. First feel for the radial pulse and if impalpable feel the femoral artery. As a last resort feel a single carotid artery. Palpate for at least one minute with a warm hand.

Blood pressure – this may be impossible to measure because of low blood pressure or because the arteries have become stiff and cannot transmit the pulse waves.

Conscious hypothermic patients may be given a sweetened hot drink, e.g. cocoa or jello. Hot food and drink will provide some heat energy and aid in rewarming but cannot be expected in themselves to add much heat to the body. The most important aspect here is the caloric intake to fuel further shivering heat production. If intravenous fluid is given, it must be warm. Initially give 250–500 ml glucose 5 % in normal saline, warmed to 38 °C. If possible, record ECG while the conscious patient is in transit. Violent shivering will make it difficult to achieve a readable tracing. **Handle the patient with care and avoid sudden movements that might stimulate ventricular fibrillation.** As the patient warms the risk of hypovolaemia and rewarming shock increases, unless there is adequate fluid replacement.

Disability – Determine the patient's level of consciousness (LOC). Patient LOC can be described by the acronym AVPU which stands for:

A – Alert
V – Responds to verbal stimulus
P – Responds to painful stimulus
U – Unresponsive

Conduct a more thorough neurologicalal examination using the Glasgow Coma Scale (GCS), as soon as possible. Even though the Glasgow Coma Scale rating is not prognostic – there is no counterpart on the neurologicalal scale for hypothermia – it must still be recorded, because the trend during a prolonged evacuation may be useful.

Glasgow Coma Scale

EYE OPENING	
Spontaneously	4
To voice stimulus	3
To pain	2
No eye opening	1

VERBAL RESPONSE	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
No sounds	1

MOTOR RESPONSE	
Follows commands	6
Localises a pain stimulus	5
Withdraws from pain	4
Flexor posturing to pain	3
Extensor posturing to pain	2
No response to pain	1

Add the scores. The lower the final score the worse the condition of the patient.

Exposure and environmental control, degrees – assess temperature. Use a thermometer able to measure down to at least +25 °C. Measure the temperature orally, rectally or in the ear with a Metraux thermometer.

In the outdoors and especially in bad weather, measuring temperature may be difficult or impossible. In an urban setting, measuring the patient's temperature is not necessary until he or she is admitted to hospital.

Two factors are involved in taking a temperature: the type of thermometer and the anatomic site for the measurement.

Electronic thermometers are best but not always reliable, especially in cold. Alkaline batteries are unreliable in cold temperatures: lithium batteries withstand cold better and last longer. It is preferable to have a thermometer that measures down to at least 20–25 °C (Reisinger et al 1979), although a standard clinical thermometer that measures down to only 34 °C may be accurate enough to determine that a victim's temperature is lower than 35 °C. Tympanic thermometers have become increasingly popular because of the ease of access of the ear and the speed and convenience of making the measurement. The resultant readings however, are not always accurate in an outdoor setting (Rogers 1999).

Measurement site: the options outside hospital are -esophageal, rectal, oral, axillary, aural.

Esophageal temperature measurement-provides the closest indication of heart temperature and can be practically measured during prehospital transport.

Rectal temperature measurement – is accurate but hardly realistic in most cases, e.g. on exposed mountain slopes.

Oral temperature measurements – are influenced by the consciousness of the patient. In an unconscious patient who has been lying prone with an open mouth, measurement will obviously be very inaccurate. In a fully conscious patient the measurement may be accurate enough, although the result may be deceptively low.

Axillary temperatures are almost always inaccurate because the skin temperature is influenced by ambient temperature.

The ear is easily accessible, and is a tempting site for measuring temperature. Devices for measuring tympanic membrane temperature are inaccurate, but use of the Metraux thermometer for measuring ear canal temperature has been proven to be reliable in the field (Walpoth 1994).

Dry – treat the patient to avoid further heat loss. Remove wet clothing with extreme care, cut the clothes off in the case of severe hypothermia. Insulate the patient with blankets, sleeping bags, bubble-wrap, and/or rescue bags.

Avoid protracted treatment in the field. First remove the patient to a sheltered area, out of wind, rain and snow. Second, protect the patient from further heat loss. Remove wet *clothing because* it rapidly conducts heat away from the body. Put the victim in dry clothes or a sleeping bag. Cover the head warmly. Insulate with extra blankets. Add extra insulation to the head and feet. The outer layer of the wrapping should be wind and waterproof. In all but the mildest cases, keep the patient horizontal until rewarmed and lucid. If circumstances do not allow the removal of wet clothing, the alternative is to leave the clothing where it is and insulate outside it. This creates a warm wet compress if external heat such as hot water bottles are placed around the victim.

There are several methods of providing exogenous heat – hot water bottles, chemical heating pads, the Heat Pac® system, warming of the forearms and lower legs (Vangaard and Gjerloff 1979, Giesbrecht 2000) and inhalation of warmed gases. While these methods might warm the patient, several of them also heat the skin and inhibit shivering (Giesbrecht 1994, 2000). Cessation of shivering may slow down natural rewarming, although the patient may feel more comfortable. If the patient is fully conscious and shivering strongly it may be more effective and quicker to dry and insulate the patient and rely on shivering to rewarm, rather than apply exogenous heat and slow down the process.

If the patient is not shivering, core temperature will likely continue to drop and stay at a low level. In this case careful provision of heat to the torso will prevent further cooling if not promote slow warming of the heart.

Unconscious patients

The unconscious, hypothermic patient is in a fragile metabolic state and may die unless rewarmed. Care is a challenge and involves total physiologic management (Mills 1992). When cold, stiff and cyanotic patients are found with fixed, dilated pupils and no apparent pulse, the basic attitude must be: "no one is dead until they are warm and dead" (Edwards et al 1970; Gregory et al 1972; Auerbach 1990). Every reasonable measure must be taken to get the patient to a hospital where full resuscitation can be initiated.

Examination of the patient must include:

A – Airway management and cervical spine control
B – Breathing (ventilation)
C – Circulation
D – Disability
E – Exposure and environmental control, degrees and dry

Airway and Breathing – Establish and maintain a free airway and adequate ventilation.

Breathing can be both slow and superficial and the diagnosis of respiratory failure difficult, but the basic rule is: if in doubt – act. If the patient is breathing, supply oxygen at a high flow, 4–6 l/min with a bridle or 40–60 % with a mask; this reduces the risk of ventricular fibrillation. The demand for oxygen in a hypothermic victim is less than in a person who is normothermic; therefore criteria for administering oxygen in hypothermia need not be the same as in normothermia, although oxygen still reduces the chances of ventricular arrhythmias, therefore administer warm, humidified oxygen if possible. Cold oxygen might reduce patient temperature still further, but is still beneficial. Do not store gas cylinders in cold spaces or directly on the ground or snow. The amount of heat supplied by warmed air or oxygen is small but can help to stabilise the patient's condition and prevent further heat loss (Lloyd 1996). Light-weight equipment is available for supplying warm, moistened oxygen in field conditions (Danzl et al 1995). According to Harnett et al (1980), administration of warm, moistened inhaled air is a safe technique to use in the field.

The indications for intubation are the same as with a normothermic victim. Oral intubation is the first preference, experience having shown that nasotracheal intubation can lead to severe bleeding from the nasopharynx. Sometimes, though, one has no choice, because it is impossible to open the patient's mouth. Take care when fixing the tube and be careful with the cuff, so that it will not break when stiffened by the cold (Dahlgren et al 1988). Intubation is considered capable of triggering ventricular fibrillation, though this is contradicted by data from a series of 428

hypothermic patients, 117 of whom were intubated by many different people. None of the intubated patients developed ventricular fibrillation (Danzl et al 1987). The fear of inducing ventricular fibrillation should not prevent intubation when this process is necessary.

Circulation

Cardiopulmonary resuscitation (CPR)

Cardiac arrest is hard to diagnose in a hypothermic patient. A pulse may not be palpable and breathing might be so slow that it is difficult to see. Chest compression to start CPR in a very cold patient with a barely perceptible heart beat can cause ventricular fibrillation and lead to the death of the patient. CPR therefore, must not be started if the patient is breathing, groaning or moving, and it must not be started if the EKG reveals isolated QRS complexes.

EKG-monitoring may turn out to be difficult. Most monitors have not been tested below 15 °C. Be prepared for the electrodes refusing to stick to the skin. Needle electrodes can be useful. EKG can be expected to show a slow frequency, wide complexes, J-wave or ventricular fibrillation. Tracing may be very small and concealed by artefacts from shivering. Asystole and ventricular fibrillation may sometimes be hard to tell apart because of low frequency tracing. A semi-automatic defibrillator can "misinterpret" the EKG and should therefore be used cautiously.

CPR must not begin until cardiac arrest has been proven, which in practice means that an EKG must be connected, and this can only be done in a sheltered place or during transit in an ambulance or helicopter. Pulmonary ventilation however, may be beneficial and can be started even if there is doubt about the action of the heart.

Many authors have recommended giving CPR at "half speed" (Danzl 1991). Although heart rate declines in linear relation to temperature, CPR should not necessarily be given more slowly than usual. Theoretically, the compression rate chosen should be that which allows the maximum filling time for the left ventricle. Admittedly, this rate may be difficult to calculate. Because of chest wall stiffness, compression may be difficult and the resulting output reduced. If CPR is begun and neither a pulse nor an EKG complex results, the treatment must still be maintained all the way to the hospital. If EKG monitoring reveals asystole or ventricular fibrillation, an attempt can be made to defibrillate with 2 Ws/kg up to 200 Ws (Tacker et al 1981; McDonald 1982). Defibrillation however, seldom succeeds below 30 °C (Lloyd and Michell 1974). Ventilation during CPR, or if assisting ventilation with sustained circulation, must be conducted at reduced speed, because otherwise a pronounced respiratory alkalosis might occur, which in turn predisposes to ventricular fibrillation.

Several hypothermic patients have survived after many hours of CPR (Gilbert 2000). Therefore, if there is no associated illness or injury that would cause death, persist with CPR until the patient is warm. If the patient is rewarmed to at least 33 ºC and there is still no cardiac response, death can be announced.

Intravenous fluids

Peripheral veins can be extremely hard to locate. Intraosseous infusion in hypothermic patients has not yet been reported, but could be a possibility when there is no alternative site. Give 250–500 ml glucose 5 % in normal saline, pre-warmed to 37–40 ºC. Equipment for heating IV fluids and tubing is essential. The temperature of exposed solutions falls very quickly, and in unprotected hoses these will freeze within two or three minutes. Cold infusions aggravate the patient's condition. Trauma combined with hypothermia makes the supply of warm fluid in large quantities imperative. Cold intravenous infusions (<37 ºC) given to trauma patients may cause hypothermia and increase the mortality rate.

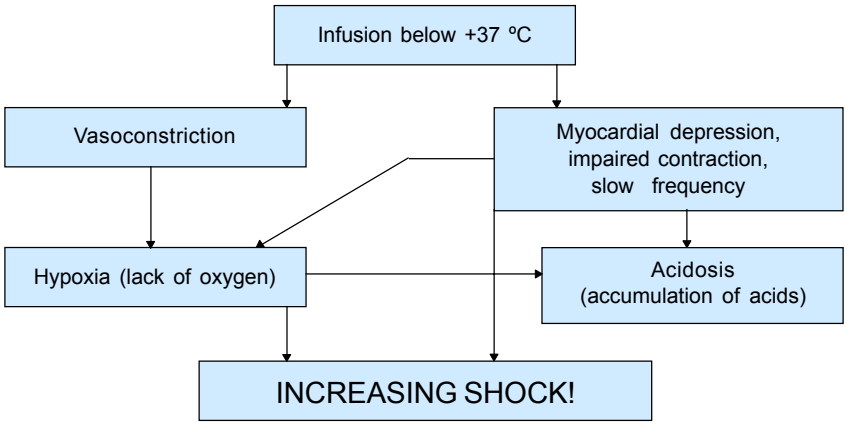


Fig 7. Infusion of cold drip solutions, <37 °C, is liable to aggravate the patient's condition (Börje Renström, personal communication)

Disability – Determine the patient's level of consciousness (LOC). Patient LOC can be described by the acronym AVPU which stands for:

A – Alert
V – Responds to verbal stimulus
P – Responds to painful stimulus
U – Unresponsive

As soon as possible make a more thorough neurologicalal examination using the Glasgow Coma Scale (GCS).

Exposure and environmental control, Degrees – Assess the temperature. Use a low-reading thermometer able to measure down to at least +25 °C. Measure the temperature orally, rectally or in the ear with a Metraux thermometer.

The ability to sustain the injured person's body temperature has to be adapted to "field" conditions. *Remove wet clothing carefully and insulate* with dry blankets, sleeping-bags, "bubble-wrap" or waterproof bags. The question of rewarming in the field has been controversial. Currently (2002) there is no effective field method available for rewarming a severely hypothermic patient. The problem of rewarming too rapidly in the field does not therefore arise. Warming in the field is directed towards preventing further heat loss, rather than actively raising core temperature. Mildly hypothermic, shivering patients may only need drying and insulation. They will rewarm themselves. A severely hypothermic patient, however, needs to be properly wrapped and protected from wind, snow and rain. If an exogenous heat source is available, such as hot water bottles, chemical heating pads or a Heat Pac®, it should be used, taking care to avoid direct contact between the skin of the patient and the source of heat because cold skin is easily burned. The need to separate the skin from the heat source reduces the heating capacity and efficiency of all these methods and makes overheating almost impossible. A traditional method of warming is to put the victim and a rescue worker together in a sleeping bag. This is a very old method but recent experimental evidence indicates that the contact may stop shivering in the victim and slow the rewarming (Giesbrecht 1994). The area of skin surface through which heat exchange takes place is limited and if clothes are worn the insulation may prevent any significant heat exchange. The method may afford comfort to the victim but should not be expected to result in rapid rewarming. In the case of a non-shivering victim however, this procedure may be better than providing no heat at all.

Patient position

During rescue, in transit and in hospital, if possible, maintain the patient in a horizontal position. This should be especially borne in mind in helicopter rescue operations, where winching up the patient in a vertical position (as shown in Fig 8) can precipitate fatal hypovolaemic shock. In certain rescue situations, however, lifting the patient in a vertical position is the only option.

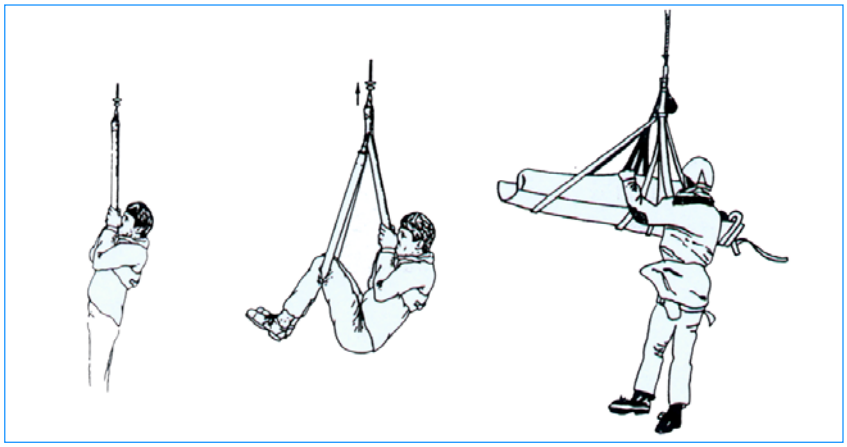


Fig 8. Horizontal lifting can save the life of the hypothermic patient

The effect of anti-shock trousers in connection with hypothermia has been poorly investigated (Kolodzik et al 1988). Hypothermic patients who already have maximum vasoconstriction are unlikely to derive much benefit from anti-shock trousers. Moreover, these trousers probably aggravate the risk of compartment syndrome, muscular necrosis and peripheral frostbite injury, and they are therefore not recommended in hypothermic patients.

Transport

Off-road transport of the hypothermic patient must be conducted with care. Whatever the mode of transport adopted, the risk of further heat loss in transit has to be avoided. Here again, the rule is to insulate the patient as well as possible, using dry blankets or rescue bags. In a helicopter, ambulance or other vehicle where heat can be generated, the ambient temperature should be 25–30 °C.

There has been a belief that no attempt should be made to warm patients during transportation because they are "safe" in a "metabolic ice box" if their core temperature is below 28 °C. However the risk of ventricular fibrillation becomes greater the lower the core temperature. It is therefore safer to have the core temperature rising rather than allow it to remain lower. If there is a cardiac rhythm there is a chance that warming may have a beneficial effect, even if only to prevent after-drop. If there is no cardiac rhythm, no warming method will be effective. The danger of causing vasodilatation and a fall in blood pressure is very slight unless the transport time is prolonged and very active warming is being done without simultaneous fluid replacement.

Place warming devices such as hot water bottles or charcoal heaters around the trunk, in the axillae and groin and fold the patient's hands on

the chest over the device. If the patient is being transported in the open on a litter, make sure the hands and feet are kept warm to avoid frostbite from exposure. Avoid direct contact between the skin of the patient and the source of heat because cold skin is easily burned. **Gentle handling, maintenance of a horizontal position, good insulation of a dry patient and respiratory ventilation are the most important considerations during transportation.** Warming the transportation vehicle will probably have little effect as the insulation around the patient will prevent significant heat transfer.

If possible, surveillance in transit should include continuous EKG monitoring. Pulse oximetry seldom works on hypothermic patients, owing to their pronounced vasoconstriction and the measurement obtained on a severely vasoconstricted finger may be meaningless. Maintenance of a free airway, ventilation and CPR conform to the guidelines already described. The patient should be conveyed to a facility offering qualified expertise and resources for rewarming under qualified supervision.

Hospital care

Initial actions and tests

A – Airway management and cervical spine control
B – Breathing (ventilation)
C – Circulation
D – Disability
E – Exposure and environmental control, Degrees and Dry

The *Airway* should be checked to ensure it is open and clear and that no danger of obstruction exists. Administer oxygen, 4–6 l/min on a bridle or 40–60 % with a mask, preferably warm and moisturised to reduce further heat loss and diminish the risk of arrhythmias (Budd 1986; Bowman 1993; Mills 1992; Lloyd 1996). If endotracheal intubation is indicated, oxygenate the patient well through a mask before attempting intubation. Indications for endotracheal intubation are the same as in a normothermic patient. Oral intubation is recommended if the mouth can be opened sufficiently to permit access. Nasal intubation may cause severe bleeding because of the danger of coagulopathy.

Monitor oxygenation: First, attach an oximeter, but be prepared for inaccurate readings due to pronounced peripheral vasoconstriction (Clayton et al 1991). Insert an arterial line both for measurement of blood pressure and for sampling arterial blood.

Insert a nasogastric tube because ventricular dilatation and poor gastrointestinal motility are common and increase the risk of vomiting and aspiration.

Cervical spine control – when checking and establishing an open airway, there is always the possibility that a cervical spine injury may exist. Thus early immobilisation is essential in most severely injured patients.

Breathing-Ventilation – Check spontaneity of ventilation. Respiration is often very slow with the respiratory rate as low as 5–10 per minute. Tidal volumes can be very small and breathing superficial. If breathing is not spontaneous, ventilation is administered.

Circulation – Monitor the EKG continuously. Evaluate the pulse for presence, quality and regularity. All pulses in the hypothermic may be extremely difficult to find. First feel for the radial pulse, if impalpable feel for the femoral artery and as a last resort feel for a single carotid artery. Palpate for at least one minute with a warm hand.

Blood pressure: this may be impossible to measure because of low blood pressure or because the arteries have become stiff and can not transmit the pulse waves. Insert an arterial line.

Establish an intravenous line percutaneously or by direct exposure. A central venous catheter is useful for assessing volumetric need, but it must not descend into the right atrium, where it is liable to trigger arrhythmias (Shields and Sixsmith 1990). Pulmonary artery (PA) catheters may also trigger arrhythmias and must be saved for complicated cases. The insertion of a PA catheter into cold blood vessels has also proved to entail a risk of pulmonary artery perforation (Pace 1977; Morris and Jande 1982; Morris et al 1985).

An indwelling urinary catheter is necessary for measuring urinary output.

Fluid therapy, mild hypothermia between 35 and 32 °C seldom requires aggressive fluid therapy, because most fluid balance changes correct themselves spontaneously when the patient begins to eat and drink. In moderate (32–28 °C) and especially in severe hypothermia (<28 °C), major fluid balance deficiencies can be expected because the patient may be significantly dehydrated (Nose 1982; Hamlet 1983). Haemoconcentration with haematocrits up to 60 %, caused by cold-diuresis and loss of fluid into the interstitial space, is to be expected. Blood viscosity increases by 2 % per °C reduction in temperature.

Electrolyte changes are to be expected but depend on the circumstances causing hypothermia and underlying associated diseases. Cold-diuresis is largely a natriuresis, leading to a low serum sodium level. Potassium levels may be either low or high. A low level is found in connection with chronic hypothermia due mainly to intracellular pooling in the muscles. Hyperkalaemia caused by a defective sodium-potassium pump

can sometimes be seen in acute hypothermia below 30 °C combined with hypoxia that leads to potassium leakage from the cells into the blood. Hyperkalaemia is also seen in connection with a simultaneous crush injury, renal insufficiency, haemolysis or asphyxiation, as in trauma or an avalanche. Hyperkalaemia in connection with severe hypothermia appears to be a poor prognostic indicator (Hauty et al 1987). Mair (1994) found that a serum potassium greater than 9 mmol/l, a pH less than 6.50 and an accelerated coagulation test (ACT) greater than 400 seconds were signs of a poor prognosis in avalanche victims in whom death preceded cooling. But after cold water submersion these same findings did not necessarily indicate a poor prognosis.

Most patients with a temperature below 32°C must initially be given 250–500 ml warm (37–40 °C glucose 5 % in normal saline) in the first 15 minutes. Ringer's lactate should be avoided, because in hypothermia the liver has difficulty metabolising lactate. Estimation of continuing fluid treatment conforms to the current standards for normothermia. Monitor in the usual way; follow the clinical picture, measure CVP, and take a chest X-ray. Avoid using a PA catheter, except for the most severe cases, until the patient has been rewarmed (Eisenberg et al 1984). A combination of hypovolaemia due to cold-diuresis and simultaneous trauma, plus increasing vasodilitation due to rewarming, can be critical (Harari et al 1975). In many cases of severe hypothermia, rapid fluid replacement can save life (Harari et al 1975; Bangs 1984; Danzl et al 1989). There is no advantage of colloid over crystalloid solutions unless there has been blood loss.

Disability – Determine the patient's level of consciousness (LOC) using the Glasgow Coma Scale (GCS). Make a more thorough neurologicalal examination as soon as possible.

Exposure and environmental control, degrees. Undress the patient cautiously (cut the clothing away) and insulate with dry, warmed blankets. Take great care to handle the patient gently during transit

Temperature measurement to confirm the diagnosis. Measure core temperature continuously. The potential anatomical sites for measurement are: oesophagus, pulmonary artery, rectum, bladder, tympanic membrane, axilla, mouth.

Oesophagus: The most accurate, least invasive measurements of true core temperature are obtained from the mid-oesophagus. This also measures left atrial and blood temperature because of the proximity of the mid-oesophagus to the left atrium. If the probe is in the upper third of the oesophagus, it comes close to the trachea and can be affected by the temperature of inhaled gases (White et al 1984; White et al 1985; White et al 1987).

Pulmonary artery: a Swan-Ganz pulmonary artery catheter may be necessary in the most severely hypothermic patients to obtain maximal physiological control. The pulmonary artery temperature can then be monitored continuously and is the most accurate and useful core temperature available.

Rectum and bladder: the rectum and bladder are the next most accurate sites. Both have been shown to reflect core temperature. Rectal temperature may fall slower than the oesophageal temperature and there may be a significant lag in restoration of rectal temperature during rewarming (Webb 1973; Edwards et al 1978; White et al 1987, Giesbrecht 2000).

The accuracy of a rectal temperature depends on the site at which the temperature is measured, A glass rectal thermometer can only be inserted 3–4 cm and the measurement is unlikely to be a true core temperature. A flexible probe, inserted at least 10 cm, and lying in contact with the bowel wall is much more likely to reflect a true core temperature. During rewarming rectal temperature can be several degrees lower than the oesophageal temperature and reliance on rectal measurements may give a false impression of the rate of rewarming. Bladder temperature is an accurate reflection of core temperature and, as many patients need an indwelling urinary catheter, the same catheter can be used for measuring temperature (Lilly 1980). The measurement of voided urine temperature is not as accurate as the measurement of temperature within the bladder and should only be used if other methods are not available (Ehrenkranz 1986).

Tympanic temperature: Measurement of tympanic temperature has become widely used because of ease of access of the ear under all circumstances and the simplicity and ruggedness of the thermometers. Temperature measurement against the ear drum correlates well with the temperature of the hypothalamus, but can be painful (Brinnel and Cabanac 1989). This has led to the development of thermometers which measure infrared radiation from the eardrum (Infrared emission detection devices: IRED) and calculate the temperature on this basis. These thermometers are only accurate under limited conditions of ambient temperature. If the ambient temperature is too cold or too hot, the temperatures recorded vary widely from simultaneously measured rectal temperatures (Rogers 1999). For these reasons IRED thermometers cannot be recommended for measuring core temperature in the field but may be more useful in the hospital environment. Most studies of IRED thermometers have involved the diagnosis of fevers in children, and they are inaccurate under these circumstances. The accuracy of their use in hypothermic patients is unproven and reliance should be placed on the use of other methods because the recorded readings may be one to one half a

degree Celsius off the rectal temperature. The Metraux thermometer that measures closed ear canal temperature, and not temperature radiated from the tympanic membrane, seems to be a more accurate method for measuring ear temperature (Walpoth 1994).

Axillary and oral temperatures are usually too inaccurate to be used in treating a hypothermic patient

Laboratory tests

Laboratory tests must include: blood-glucose, blood gas, blood count, electrolytes, liver function tests, cardiac markers, including CK, CKMB, and Troponin-I or Troponin-T, coagulation status, urea, magnesium, and amylase. Consider a toxic screen if there is any suspicion of drug ingestion.

X-ray examination: Use appropriately if traumatic injuries are suspected. Pulmonary X-rays provide useful guidance for volumetric treatment, for confirming the position of a central venous catheter, and to diagnose pneumonia and ARDS. Computer tomography and ultrasonic examination of the abdomen can be considered in trauma cases, because clinical assessment of the abdomen is made difficult by a cold, stiff, anaesthetic abdominal wall.

Rewarming

Before rewarming begins in hospital, the patient must be under complete monitoring and have functioning intravenous lines. As rewarming proceeds, EKG, blood gas, electrolyte status and blood glucose must be kept under close observation.

Passive rewarming

The borderline between the need for passive and active rewarming usually coincides with a body temperature of 32 °C. In the 35–32 °C range, drying the patient, insulating with blankets or the equivalent in warm surroundings (room temperature 25 °C) and administration of warm drinks and calories are recommended. The rewarming rate will be 0.5–2 °C/hour (Maclean and Emslie-Smith 1977; Danzl 1983; Danzl et al 1987; Danzl 1988). In situations where qualified resources are lacking (see below) and in connection with disasters, this is the method which has to be used in patients even with body temperatures below 32 °C.

Active rewarming

Active rewarming is necessary when the core temperature is lower than 32 °C. Heat can be administered externally or internally, direct to the body core.

External rewarming

In active external rewarming heat is supplied through the patient's skin (Fernandez et al 1970; Meriwether and Goodman 1972). There are many variants of this, e.g. hot-water bottles, thermal blankets circulating warm water and forced air blankets that blow warm air onto the patient (e.g. Bair Hugger) and thermal ceilings, hot water immersion, and warming the venous return from the arms and legs (Vangaard technique). The equipment for all these methods is normally available in modern intensive care units. All warming devices in direct contact with the skin should be used with great caution as cold skin is easily burned (Crino and Nagel 1968; Cohen 1985; Feldman et al 1985). External rewarming as described above, (with the exception of the Vangaard technique, see below) should initially be limited to the trunk, so as to raise the temperature of the core of the body above the arrhythmia level (28–32 °C) as quickly as possible.

Forced air warming The Bair Hugger, designed for rewarming patients after surgical operations, has proved to be an effective and atraumatic method of rewarming hypothermic patients (Brauer et al 1999; Kornberger 1999; Deakin 2000). A portable model has been designed that may be useful in ambulances and rescue helicopters (Giesbrecht et al 1998).

The Vangaard technique, used in the Royal Danish Navy (Vangaard and Gjerloff 1979) makes use of a different theory, that by warming the lower legs and arms to the knees and elbows, the superficial veins can be used as heat exchangers. The method is only suitable for conscious patients. Both legs to the knees, and both arms to the elbows are immersed in 45 °C water. It is important that sufficient areas of all limbs are immersed and that the water be warm enough. Experiments have shown that rewarming rates as high as 9.9 °C/hr can be achieved (Giesbrecht 2000).

A hot water bath is capable of rewarming both periphery and the core. Bath rewarming requires immersion of the whole of the patient's body, except the head, in water at a constant temperature of 40–42 °C (Hoskin et al 1986). Establish intravenous lines before putting the patient in the bath. EKG monitoring can be performed with telemetry or with standard EKG leads using Nobecutan sprayed plates or needle electrodes. The patient is placed on a vertically adjustable stretcher and then lowered into the water. Immediately before the patient is lowered into the

bath, 500 ml warm (37–40 °C) glucose 5 % in normal saline is infused relatively quickly. As the peripheral blood vessels dilate in the warm water, a further 500 ml is administered. Once a stable blood volume has been established the fluid infusion can be slowed. If ventricular fibrillation develops, the patient must be lifted out of the water and dried before defibrillation . The warm water opens the peripheral circulation. The superficial vessels along the arms and legs will absorb copious amounts of heat which are transmitted directly to the heart. Thus both external and internal rewarming will be achieved. From a circulatory viewpoint, the hydrostatic pressure in the bath will bring about some compression of the superficial blood vessels, thereby boosting the venous return. An anti-shock position is important, however, to avoid hypotension. Rewarming progresses rapidly, taking between 45 and 120 minutes. Rewarming in a bath makes CPR and defibrillation difficult, therefore a carefully trained staff is needed in order for the rewarming model to work safely (Frank and Robson 1980). Despite these objections, Dr Mills of Anchorage has been using the method successfully for many years (personal communication). As the immersion model presents difficulties, in as well monitoring as resuscitation, we recommend the method to be used with great care and only in mild to moderate hypothermia and when the patient is conscious.

Internal rewarming

Several methods have been described: inhalation of warm, moistened air, peritoneal lavage, thoracic lavage, haemodialysis and extra-corporal circulation.

Inhalation of warm air has been described for use both in the field and in hospital (Moritz et al 1945; Wessel et al 1966; Romet and Hoskin 1988; Lloyd 1991, 1996). Dry air, because it is a poor carrier of heat, has to be humidified. Inhalation temperature must be between 40 and 45 °C. The rewarming rate is greater if the patient is intubated than if administration is by mask. A 40 °C aerosol gives a rewarming rate of 0.7 °C/hour with a mask and 1.2 °C/hour with intubation (Miller et al 1980; Miller et al 1981). The rate of rewarming corresponds to that of passive external rewarming and is thus a slow method. Therefore it is not recommended as the sole rewarming method, but should be combined with other methods. It does however, have attractive advantages. Warm humidified air/oxygen assures the patient of adequate oxygenation, stimulates cilial motility and reduces the quantity and viscosity of cold-induced bronchial mucus (Morrison et al 1979; Morrison et al 1982; Linko et al 1984). Many commercial humidifiers require modification to reach the 40–45 °C desired. If modified, they must be clearly labelled so that they will not be used for routine purposes and cause hyperthermia.

Wallace (1997) has raised a warning about overheating the inspired gases and causing damage to the respiratory tract.

Peritoneal lavage is an efficient, safe method available in most hospitals. Heat is administered intraperitoneally by perfusion with an isotonic dialysis fluid at a temperature of 40–45 °C (Klarskov and Amter 1976; Soung et al 1977; Reuler and Parker 1978). This method allows simultaneous detoxification in cases of drug ingestion. Both hypokalaemia and hyperkalaemia can be treated by appropriate manipulation of the dialysis fluid.

There are two safe methods of performing peritoneal lavage. The best, under local anaesthetic, is to expose the peritoneum through a 1–2 cm long incision below the umbilicus, then the lavage catheter is inserted, under visual control, through the peritoneum towards the true pelvis (Moore et al 1981; Moore et al 1991). By the second method, a semi-open technique, dissect down to the fascia in the midline and then pass a needle through fascia and peritoneum through which a guide wire is introduced. A lavage catheter is then passed over the guide wire (Seldinger technique). The closed method, using blind perforation of the abdominal wall, has a high complication rate and is not recommended for these cases. If the patient has had a previous abdominal operation, use an open technique to avoid damaging intraperitoneal structures.

The dialysis fluid can be isotonic sodium chloride solution or a dialysis fluid with glucose added. The fluid is heated to 40–45 °C and up to 2 litres are infused, left for 20 or 30 minutes and then aspirated or allowed to drain out under gravity. Normally an exchange of 6 l/hour can be achieved, which provides a warming rate of 1–3 °C/hour. Double catheter systems with output suction accelerate the rewarming rate (Grossheim 1973; Johnson 1977; Pickering et al 1977; Bristow 1978; Jessen and Hagelsten 1978; Desmeules and Blais 1979; White et al 1985). Danzl et al (1995) recommend using a larger catheter, such as an Arrow 14 Fr drainage catheter, which accelerates the drainage exchange. This form of rewarming should be feasible at most hospitals. It also permits simultaneous CPR, should the need arise.

Thoracic lavage : Rewarming with warm fluid in one or both pleurae has been used in treating patients with post hypothermic cardiac arrest (Brunette et al 1987, 1992; Hall 1990; Kangas 1994). Two relatively large chest tubes, (36–40 Fr for adults, 20–32 Fr for children aged 4–7, 14–24 Fr for children aged 1–3) are inserted in one or both pleurae. One is placed anteriorly in the second to third intercostal space at the mid-clavicular line, the other in the posterior axillary line through the fourth or fifth intercostal space. One to three litres of normal saline, heated to 40–42 °C, are then infused through the superior drain. The aid of a high-flow heat exchanger can assist the infusion. Drainage takes place spontane-

ously through the inferior drain and can be collected in a standard chest drainage system. The collecting vessel has to be emptied frequently and has to be working properly, to avoid a tension hydrothorax. If you can choose between sides, infuse through the left pleura as a greater mass of the heart will be exposed to the warm water than if the infusion is on the right side.

Haemodialysis: The availability of two-way flow catheters which can be inserted percutaneously, through the internal jugular or the subclavian veins, has made haemodialysis a more readily available rewarming technique. Exchange cycles of 200–250 ml/min can result in moderately rapid rewarming. Haemodialysis can often become necessary as an adjunct to other rewarming techniques for hyperkalaemia, intoxication and muscular injury (Myers et al 1979; Laub et al 1989; Tan 1990; Hernandez 1993, Danzl, 2001).

Extracorporeal circulation: Cardiopulmonary bypass has become a standard treatment for severe hypothermia. The great advantage of this method is the ability to maintain an oxygenated blood flow, even in the event of cardiac arrest during rewarming (Davies et al 1967; Kugelberg et al 1967; Dorsey 1980; Caldwell et al 1981; Husby et al 1990; Husby et al 1991; Tisherman et al 1991; Deimi and Hess 1992; Jones and Swann 1994; Vretenar 1994; Mair 1997; Walpoth 1997).

Cardiopulmonary bypass may be either partial or complete. Complete bypass is established by a midline sternotomy, cardiac cannulation and connection to a heart-lung machine. The standard partial model is femoro-femoral bypass including arterial and venous catheters, a mechanical pump, membrane or bubble oxygenator and a heat exchanger (Bolgiano et al 1992; Walpoth 1997). A 16–30 Fr. venous catheter is inserted in the femoral vein and moved up to the transition between the inferior vena cava and the right atrium. A second 16–30 Fr arterial catheter is passed up the femoral artery to the aorta bifurcation. Flows of 2–3 l/min can raise the temperature by 1–2 °C every five minutes. The usefulness of this technique in association with simultaneous trauma, is limited by the necessity for full heparinization. New methods with reduced anticoagulation have been developed (Gravlee 1994; Irone et al 1998). Successful treatment without systematic heparinization but with heparin-coated perfusion catheters, has been performed in patients with a body temperature of 23 °C, cardiac arrest and simultaneous intracranial trauma (von Segesser et al 1991; Wang et al 1991; von Segesser et al 1992). Complications of this technique include vascular injury, air embolism, haemolysis, ARDS (Adult Respiratory Distress Syndrome) and pulmonary oedema. Endothelial leakage augments the risk of compartment syndrome and peripheral oedema. The report from the Mt Hood accident in 1988 contains dramatic examples of these complications (Wilkerson and Hamlet

1988). Patients resuscitated with extracorporeal circulation require particularly close physiologic monitoring because of the danger of major fluid overload resulting in pulmonary or cerebral oedema.

Other conceivable models for extracorporal rewarming but without the possibility of oxygenation, are the use of arterio-venous or veno-venous circuits over a counter-current fluid warmer (O'Bryne et al 1989; Gentilello and Rifley 1991; Gregory et al 1991; Gentilello et al 1992). The cannulae are inserted percutaneously and circulation is maintained by the patient's own blood pressure which should exceed 60 mm Hg.

Resuscitation

Cardiopulmonary resuscitation

CPR with external cardiac massage can be life-saving in hypothermic patients with cardiac arrest. Cardiac arrest, however, can be hard to diagnose in hypothermia. A premature decision to start CPR in a patient with barely detectable cardiac action can cause ventricular fibrillation and cause the death of the patient. **CPR must not begin until cardiac arrest has been confirmed,** which in practice requires EKG evidence of asystole or ventricular fibrillation, since pulse, blood pressure and respiration may be impossible to measure. CPR must not be started if the patient is breathing, groaning or moving, or if the EKG reveals isolated QRS complexes. The optimum frequency and technique for CPR in connection with hypothermia are unknown. Myocardial compliance in hypothermia can be greatly reduced. Althaus et al (1982) noted during thoracotomy in a survivor that "the heart was found to be hard as stone and it is hardly conceivable how effective cardiac massage could have been". Thoracic and lung compliance are both reduced (Deal et al 1970; Edelman 1974). Thus greater effort than normal has to be exerted to compress the chest (Voorhees et al 1980; Weisfeldt et al 1981). In spite of very difficult conditions, there are many descriptions of neurologicalally intact survivors after prolonged external cardiac massage (Kukarni and Thomas 1999). In one multi-centre study of 428 hypothermia cases, 9 out of 27 survived with CPR begun in the field and 6 out of 14 survived when CPR began in the emergency department (Danzl et al 1987). One hypothermic patient survived after 6.5 hours' external cardiac massage (Lexow 1991) and another patient survived after 9 hours of resuscitation (Gilbert et al 2000). The compression rate varied from half to normal frequency.

Open airway

Assure an open free airway as for a normothermic patient (Carden 1983; Gillen et al 1986; Danzl et al 1989). The earlier debate concerning the risk of triggering ventricular fibrillation with endotracheal intubation has to a great extent been resolved. In one multi-centre study with 117 intubations, no arrhythmias were triggered (Danzl et al 1987). In another study of 40 nasotracheal intubations there were, again, no arrhythmias (Meriwether and Goodman 1972; Danzl and Thomas 1980). The risk of initiating an arrhythmia is greatly reduced if the patient is well oxygenated before intubation is attempted. Oral intubation is especially recommended in patients with coagulation effects, to avoid severe nose bleed. "Our experience is that these patients bleed like crazy from damage to the nasal mucosa" (Wilkerson and Hamlet 1988).

Sometimes, however, due to masseteric spasm induced by the cold, the patient's mouth cannot be opened, in which case blind nasal or fibre-optic nasal intubation is recommended rather than cricothyroidotomy (Danzl et al 1995). If muscle relaxants are used in connection with intubation, be prepared for a longer duration of respiratory assistance (Mazala et al 1988; Rodrigo and Ranwala 1988).

Pharmacologicalal aspects of resuscitation

Pharmacologicalal effects are generally temperature-related: the lower the temperature, the stronger the protein binding, the slower the action of drugs. Enterohepatic circulation and renal secretion are impaired resulting in slower metabolism of the drug. Abnormal drug actions are to be expected. One common clinical scenario is a diminished effect during hypothermia progressing to toxicity after rewarming (Menard and Hahn 1991). Pharmacologicalal stimulation of pulse, blood pressure and "respiratory drive" is not indicated. Vasopressors are arrhythmogenic and cannot increase peripheral resistance if the latter is already maximal (Kugelberg et al 1967). A tachycardia out of keeping with the temperature must prompt consideration of hypovolaemia, hypoglycaemia or intoxication. If the blood pressure is so low that it does not agree with what is expected at the temperature in question, and the patient is not responding to crystalloid, a low dose of dopamine (1–5μg kg/min) is primarily recommended (Oung et al 1992). Steroids are not indicated for hypothermia, unless previous history or prolonged exhausting exposure suggest adrenal cortical failure. Naloxon produces a positive effect in hypothermia caused by drug overdose and in connection with spinal shock (Holoday and Fadel 1980; Glick and Guido1982). Small doses of bicarbonate are indicated if the arterial pH is <7.20, starting with 50 ml. Otherwise there is a serious risk of overshoot and alkalosis after rewarming. "A little less, a little later" (Danzl, personal communication).

Complications during resuscitation

Atrial arrhythmias are common when the temperature falls below 32 °C (O'Keefe 1977; White 1982; Okada 1984). Atrial fibrillation usually stops spontaneously when the patient is warm, and digitalisation is not recommended (Nahun and Phillips 1959; Szekely and Wynne 1960; Beyda et al 1961).

Ventricular arrhythmias. Transitory ventricular arrhythmias are to be ignored (Rankin and Rae 1984). They are usually benign and seldom turn into ventricular tachycardia (VT) or ventricular fibrillation (VF).

Of group 1A, antiarrhythmics, procainamide has proved to increase VF frequency in the 25 to 30 °C -interval (Dundee and Clarke 1964). In group 1B, lidocaine has not shown any certain prophylactic effect, nor has it proved an effective means of facilitating defibrillation (Angelakos 1959; Nielson and Owman 1968). In group III, bretylium tosylate has been proved to be extremely effective in animal experiments. Two cases of chemical ventricular defibrillation after infusion of 10 mg/kg bretylium tosylate in a patient with accidental hypothermia are described (Danzl et al 1982; Kochar et al 1986). Amiodaron, also a group III antiarrhythmic, is a conceivable alternative to bretylium tosylate, but has not been evaluated in hypothermic conditions. Magnesium sulphate administered in a dose of 100 mg/kg intravenously has been described as spontaneously defibrillating most patients at 30 °C in a series with induced hypothermia (Buky 1970). Treatment of bradyarrhythmias with transvenous pacing is considered extremely hazardous, with a serious risk of VF. External pacing has been recommended (Motin et al 1973; Pace 1977; Faller and Rauscher 1978; Falk et al 1983).

Sepsis

Hypothermia may result from infection, and infection may follow hypothermia. Sepsis, especially gram negative sepsis, is one of the diseases that commonly leads to hypothermia. The classical signs of infection – fever and erythema – do not occur during hypothermia. Trembling can easily be mistaken for shivering or may not occur at all. The leucocyte count is normal or low, owing to bone marrow depression and sequestration in the liver and spleen (Biggar 1983). Neutrophil dysfunction has also been described (Clardy et al 1985; Sung et al 1985). Hypothermia impairs the body's resistance to infections, allowing severe bacterial infections to develop with minimal inflammatory response (Lewin 1981). The presence of hypothermia in a patient with severe sepsis and positive blood cultures indicates a poor prognosis (Pittet 1996).

In one study of 51 hypothermic children, 27 were septic (Dagan and Gorodischer 1984). The dominant pathogens were staphylococci, streptococci, haemophilias and enterobacteria. Although hypothermia is com-

mon in septic children, mortality is related to the severity of the sepsis and not to hypothermia (Sofer 2000). Long-term results in children who survive are good: development and growth are normal.

In a study of elderly hypothermic patients by Darowski (1991), the majority had a suspected or diagnosed infection. In both the old and the young, prophylactic antibiotic treatment is recommended. A combination of aminoglycoside and ampicillin has been recommended (Darowski et al 1991). Infection rates of between 1 and 40 % are described among adults (Lewin et al 1981; White 1982; Morris et al 1985; Danzl et al 1987). Prophylaxis is not recommended for this age group, but treatment is recommended if aspiration, lung infiltrate or bacteruria are suspected or difficulty is experienced in rewarming the patient. The choice of antibiotics depends on the cultures obtained and the bacteriologic diagnosis.

Results of treatment

The lowest recorded temperature of a child surviving accidental hypothermia is 15 °C, and in an adult it is 13.7 °C (Gilbert 2000). In induced hypothermia, the corresponding figure is 9 °C (Niazi and Lewis 1958; DaVee and Reineberg 1980; Nozaki et al 1986). Thousands of children have been cooled to 15.0 °C during cardiac surgery with a normal neurologicalal outcome, and the induction of total body hypothermia to 25 °C in adults is now routine practice during many cardiac operations.

Survival is hard to predict, due to the pronounced variability of man's physiological response to hypothermia. In a multi-centre study, neither age nor sex has been shown to have any firm connection, with mortality (Danzl et al 1987). In a regression analysis of 234 cases in Swiss hospital departments (Locher et al 1991), the most negative prognostic factors were: asphyxia, slow temperature loss, invasive rewarming, asystole, pulmonary oedema and development of ARDS. Positive prognostic factors are rapid heat loss, ventricular fibrillation instead of asystole, and drug or alcohol intoxication.

In a retrospective study of avalanche victims, Schaller et al (1990) found that hyperkalaemia could be a negative prognostic factor. Mair (1994), however, found an elevated serum potassium to be of variable prognostic value.

The fatal cases in the Mt Hood accident were severely hyperkalaemic, >10mmol/l (Hauty et al 1987; Wilkerson and Hamlet 1988). In both these studies, however, hyperkalaemia may have been partly due to asphyxia and crush injuries. Another explanation may be that the victims were in fact already dead when the samples were taken. In a study of mountain and submersion accidents, Kornberger (1994) found that a high plasma potassium, a low pH and a prolonged ACT were strong predictors

of a bad outcome in avalanche victims who had probably died before they became cold, although these same factors were not predictive in people who first became cold and then suffered cardiac arrest. These findings might confirm that the high potassium levels found in the Mt Hood victims were evidence of death rather than a poor prognosis. A single high potassium level on admission to the emergency department should not be taken as evidence of a hopeless outcome, but should be analysed in relation to all the other factors involved in the case – duration of hypothermia, other evidence for probable death, and core temperature.

The difficulty of forecasting the outcome of hypothermia remains. A "hypothermia outcome score" was developed from a wide hypothermia database (Danzl 1989). In practice, this has not proved to be of value. For practical purposes these words still hold true: "No one is dead until they are warm and dead." But as has been pointed out (Auerbach 1990) "In reality, some victims are clearly dead when they are cold and dead, and it would be nice to safely identify them."

The Future

A successful result depends on the co-ordinated efforts of many people – the rescuers, ambulance personnel, the staff of the emergency department and intensive care. Rapid communication, exchange of information, safe transportation and knowledgeable handling of unstable victims are all essential. As these links in the chain improve, we can anticipate more victims surviving lower temperatures with fewer complications and lower mortality.

Local cold injuries

General hypothermia and local cold injuries often occur simultaneously.

Local cold injuries can be divided into two main groups: frostbite and non-freezing cold injuries (injuries occurring in connection with cold, damp and prolonged immobilisation). Frostbite is classed as frost-nip, superficial and deep. Non-freezing cold injury (NFCI) has previously been called "trench foot" and "immersion foot"

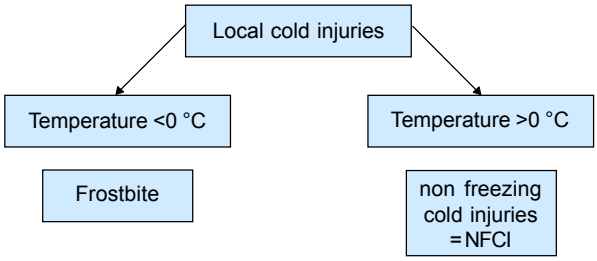


Fig 9. Freezing and non freezing cold injuries.

Epidemiology

All through history, local cold injuries have followed in the footsteps of war. During the First and Second World Wars, the Korean, and Falkland Island Wars, injuries caused by cold were massive (Paton 2000). Troops in the Falklands War, for all their modern equipment, were unable to protect themselves against cold-wet injuries of the lower extremities. Non-freezing injuries (NFCI) occur after prolonged exposure (for days) to cold and damp, coupled with immobilisation. Local cold injuries among civilians are caused by acute exposure to temperatures below freezing, resulting in frostbite. Reports of frostbite among mountaineers (Foray 1992) snowmobile drivers (Schissel 1998; Nissen 1999;) skiers (Smith 1996) and hunters are common (Knize et al 1969; Mills 1973; Mills 1975; Mills et al 1987; Ervasti et al 1991; Hashmi 1998; Catermole 1999; Conway and Husberg 1999). In civilian life the majority of cases occur in cities, frequently to the homeless, the psychiatrically disturbed and alcoholics (Valnicek 1993; Pinzur and Weaver1997; Murphy et al 2000). In recent years the inhalation of various gases by young people and the inhalation of various gases by people involved in industrial accidents have been reported as causes of freezing due to rapid evaporation and cooling (Kernbach-Wighton 1998; Kuspis and Krenezlok 1999; George 2000).

Frostbite

Etiology

The essential pathological feature of frostbite is freezing of the tissues and a wide variety of circumstances can cause freezing – exposure to severe wind-chill, (Osczevski 1995, Danielsson 1996; Candler and Ivey 1998) exposure to cold metal or water (Knize 1969), inhalation of chemical gases (George 2000). Any agent or environment that results in rapid loss of heat from the extremities and skin, whether by conduction, convection or evaporation can cause frostbite.

Danielsson (1966) found that as the skin temperature falls from -4.8 to -7.8 °C the risk of frostbite increases from 5 to 95 %. At an air temperature warmer than -10 °C there is little chance of frostbite, but at all temperatures colder than -25 °C there is considerable risk (Danielsson 1996).

Clinical picture

Frostbite often begins with a stabbing local pain, which, however, rapidly gives way to complete anaesthesia. The injury can easily be overlooked, because the pain is not very severe. The skin is pale white. At this stage the injury is superficial and can easily be thawed out by rewarming with an ungloved hand or by putting the frozen hand or foot in a comrade's warm armpit. If the injury is left untreated, the skin hardens and ice forms in the tissues. The injury then becomes deeper.

As the deep injury thaws, which is usually very painful, the skin flushes. The skin then becomes oedematous and large fluid-filled blisters appear in a matter of hours. The content of the blisters is of prognostic importance. Blisters filled with clear serum and located distally on the digits generally indicate an injury that will recover without tissue loss. Blood-filled, proximal blisters, indicate deep, severe damage and potential loss of tissue. Blisters persist for between four and ten days, after which their content is resorbed and they sometimes break spontaneously. When they dry out, a hard, eschar forms which is often black and can easily be mistaken for gangrene. The eschar separates spontaneously three or four weeks after the injury, often revealing healthy epithelium beneath. When the injury is so deep and severe that the cells die, the tissue does not flush when rewarmed but remains cold and cyanotic after thawing (Orr and Fainer 1952). No blisters appear the dead areas, most often on the distal phalanges, start to mummify within a few days. After an interval as long as three months a line of demarcation eventually appears between healthy and dead tissue. Spontaneous amputation would occur if the part were left alone. The area of black, mummified skin may be greater than the area of underlying gangrene, and the amount of tissue lost may not be

as much as anticipated. Infection, premature debridement or amputation can lead to unnecessary tissue loss, osteomyelitis and a higher amputation level than necessary (O'Malley et al 1993).

Pathophysiology

There are two important phases in the development of frostbite: damage during cooling and damage during rewarming.

Cooling: Exposure to cold causes vasoconstriction, followed by vasodilatation for four or five minutes; the 'hunting' response of Lewis (1930). As tissue temperature falls, blood is shunted past the distal capillaries (Vangaard 1990). Continuing temperature reduction leads to the extracellular formation of ice crystals that damage cells. Meryman (1956) found that large ice crystals formed by "slow" freezing, such as is experienced clinically, are more damaging to cells than "rapid" freezing. The formation of extracellular ice crystals results in an increase in osmotic pressure that leads to intracellular dehydration (Meryman 1971). The increase in extracellular concentration of electrolytes and protein degradation products progresses, intra- and extracellular pH disturbances occur, and intracellular enzyme degradation follows. Finally, the cell membrane and the sodium-potassium pump are damaged, causing greater cell wall permeability and a risk of additional cell damage (Mazur 1970). Cell damage leads to impaired microvascular circulation and capillary endothelial damage. In severe, deep frostbite nerves, muscle and bone may all become necrotic.

Rewarming: during the cooling phase there is stasis of the microcirculation resulting in tissue ischaemia. While the tissue is cold the damage is not apparent. After warming, a reperfusion injury develops. The capillary endothelium has been damaged and neutrophils adhere to the damaged cells. Activation of these neutrophils leads to the release of free oxygen radicals, thromboxane A_2 and prostaglandins and this initiates vasoconstriction and clotting (Heggers 1987) There are significant alterations in platelet function (Zook et al 1998).

Treatment of frostbite injury

The old treatment of rubbing with ice or snow recommended by Baron Larrey (O'Sullivan 1995) and followed for more than 100 years is no longer acceptable. During and after World War I ideas began to change, but it was not until the work of Ariev (1955) in Russia in the 1930's, and Fuhrman (1947) in the United States, that the benefit of rapid rewarming was proved. The first large clinical study series with patients was published in 1961 by Mills, and since then rapid warming in water, 40–42 °C has, with modifications, become the accepted treatment (Mills et al 1998).

The following case history, presented with kind permission of Dr William J. Mills Jr., is illustrative of the sequence of the events that can be anticipated in typical severe frostbite treated by rapid rewarming in water

> This fourteen year old boy went hunting, walking twelve miles during his trip. The ambient temperature was -29 °C, with little wind. He was wearing borrowed tight leather boots, covered with overshoes. One hour after leaving home his feet were noticeably cold and painful. Three hours later sensation was absent completely, the feet painless, and he continued hunting without pain, and therefore more comfortable. Six hours later he arrived home, overshoes filled with snow, and frozen to the boots. Foot gear was removed with difficulty, exposing cold rigid, pale, yellow-white discoloured feet, "solid to the ankle". He was taken to the nearest hospital. Examination there demonstrating an anaesthetic foot, digits still in the "frozen" state, with collapse and compression of the volar pads. Forty-five minutes had elapsed since removal of the shoes and by this time a purple-red line of discoloration had developed at the line of demarcation between rigid and softer proximal tissues. The area involved was without sensation or motion.
>
> The feet were thawed in water, temperature 43 °C until flushing of the distal pads was evident. The resulting "burgundy wine" hue of the distal foot persisted until the gradual development of blebs. His course was satisfactory, (Fig 10–13) and he was discharged from the hospital for home care three weeks after injury. This was a rapid course of hospital treatment for what was probably a severe injury. In our opinion this cure could only have been effected using rapid rewarming techniques.

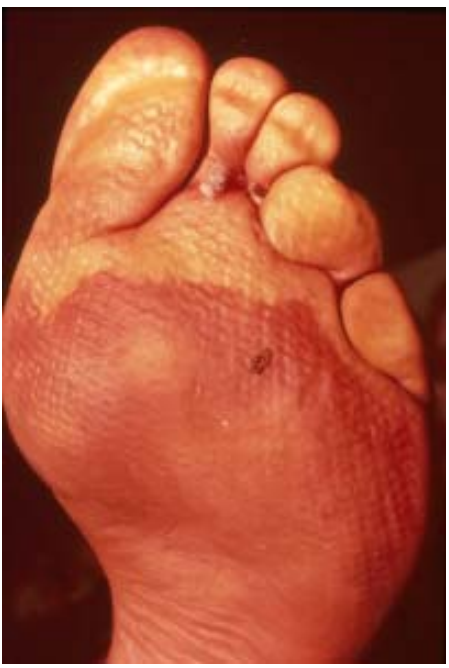


Fig 10. A cold rigid foot without sensation or digital motion. Tissue compression and sock marks are obvious. Treatment comprised a whirlpool bath and thawing at 43 °C for approximately twenty minutes.

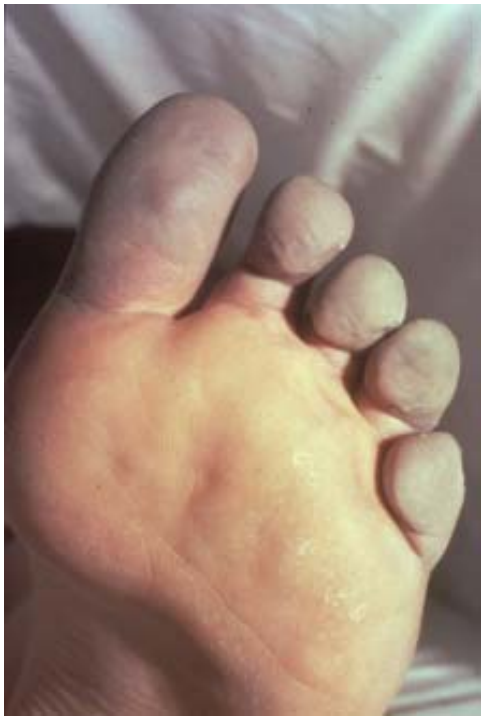


Fig 11. Thawing was followed by an ominous burgundy hue. This has since been witnessed in other cases, more often at temperatures greater than 43 °C. The cyanosis remained for approximately six hours at which time small discrete blebs began to appear. Gross sensation was present after thawing and remained so until bleb development.

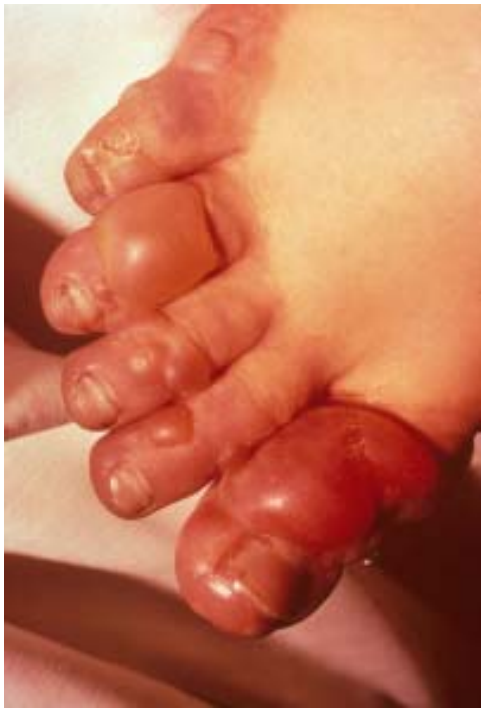


Fig 12. Over the next forty-eight hours large clear blebs develop, ultimately extending to the digital tips. Failure of distal bleb formation, in the presence of proximal blebs, is an ominous prognostic sign.

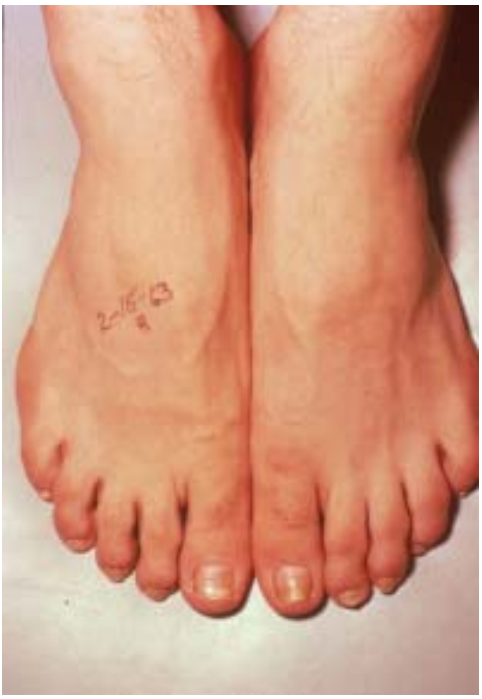


Fig 13. Four month post-injury, the anatomy has been preserved but the changes of deep injury are obvious, and include volar fat pad loss, subcutaneous fat loss, early interphalangeal joint contracture, nail changes, hypesthesia and hyperhidrosis. Epitheliazation is complete. At the end of one year the extremity had adequate sensation, there was mild subcutaneous loss and interphalangeal contracture, with a few interphalangeal subarticular lesions present on X-ray examination. Sweating increased.

Pre-hospital treatment

Superficial frostbite

The skin can still be moved over the underlying tissue. Warm the patient as soon as possible, look for shelter, provide warm clothing and administer warm drink and food. Rewarm the cold injury with your own or another person's body heat. A warm hand against a frozen nose, cheek or chin can reverse the course at an early stage. A frozen hand or foot can be rewarmed by putting it under the clothing of another person, in their axilla or on their abdomen.

Deep frostbite

The skin is immobile, stiff, white or pale bluish-grey in colour and anaesthetic. The joints may also be stiff and immobile: a hand becomes a stiff claw. Loosen tight-fitting clothing in the vicinity of the injury. Protect the frozen part from further heat loss and physical damage. Carefully remove wet clothing or shoes and insulate with dry socks or blankets. Avoid further injury by rough handling of the frozen part. Give the injured person a warm, sweetened drink. Do not permit the patient to smoke or chew tobacco. Carefully convey the patient, still thoroughly insulated, to hospital.

Difficult decisions sometimes have to be made in managing a frost-bitten limb in the outdoors. If help is going to be slow in arriving and transportation will be difficult, it may be safer to keep the part frozen rather than permit it to thaw and risk a re-freeze. The freeze-thaw-refreeze cycle is guaranteed to result in severe damage. If, for instance, a mountaineer reaches a camp with a frozen foot, removing the boot and allowing the foot to thaw will result in a swollen foot that can neither be returned to the boot, nor walked on. Frozen parts should not be intentionally thawed outside a hospital unless safe, protected transportation is assured.

Before thawing, administer ibuprofen 600 mg as soon as the patient is seen and repeat every 6 hours if transportation is prolonged.

Hospital treatment

Thawing

- Protect the frozen part of the body from mechanical trauma. Irreversible injury can result from damage to the borderline zone between damaged and undamaged tissue. Do not massage or bend the digits!
- Thaw the frozen part in water in a whirlpool bath or bath tub at a temperature of 40–42 °C.
- During thawing, it is an advantage for the person to be fully immersed in the bath. An ordinary bathtub can be used, although commercially available therapeutic bath tubs with lift, thermostat, large flows and constant water circulation are preferable.
- The viable part of the limb usually flushes within 30 minutes. Any part of the extremity that has not flushed by this time is probably not viable.

At Providence Hospital in Anchorage, Alaska, Hubbard or Arjo baths are used in which the patient is completely immersed in water at a temperature of 40–42 °C. This method is used both for peripheral cold injuries, hypothermia and combinations of both, with good results (Mills personal communication).

After thawing

- Administer ibuprofen 600 mg every 6 hours for five days.
- If the injury is superficial and only redness remains after thawing, the patient can be discharged. If the injury is more serious, with pain, blisters and swelling, the patient can still be treated on an outpatient basis, but hospitalisation must be considered.
- In cases of more extensive injury, blood blisters or poor or non-existent peripheral pulses, the patient must be detained.
- Avoid mechanical trauma and pressure against the injured part of the body.

- Nurse the patient on clean, not necessarily sterile, sheets. In the early days elevate the part to avoid swelling. Protect the part from the sheets with a cage.
- If swelling and pain are severe after warming, suspect a compartment syndrome. Measure compartment pressures. If pressure is high, a fasciotomy is performed.
- Clear, serum filled blisters can be un-roofed and treated with sterile aloe vera ointment, a mild prostaglandin inhibitor. Blood filled blisters should be left intact and allowed to shrivel naturally. If they burst, dress them with antibiotic ointment.

The injured limb is treated in a whirlpool bath, twice daily at 40–42 °C, 20 minutes at a time to debride loose tissue and remove superficial bacteria. Add chlorohexidine or Alsol® solution to the bath. Early during the course, technetium scintigraphy or triple phase scanning (Greenwald 1998) should be performed in order to assess the location of the distal limit of viability. This has proved to be a good, dependable method of forecasting the extent of damage (O'Malley and Mills 1993). Magnetic resonance scanning [MRI] and magnetic resonance angiography [MRA] also provide good assessment of circulation in the frozen area soon after injury (Barker 1997).

Amputation should be delayed until enough time has passed for demarcation to become distinct, at least 21–45 days. If there is infection, limited amputation may be necessary early in the treatment.

In cases of combined fracture and frostbite, stop external bleeding, rewarm as soon as possible and preferably set and cast the fracture without operation. The total fluid balance and nutritional state of the injured person must be assessed. In the event of dehydration, administer warm IV fluid. Provide a high-protein, high calorie diet. Antibiotics are not necessary in the absence of obvious infection. If the patient has an avascular extremity, watch for the development of sepsis. If there are signs of sepsis, wide-spectrum antibiotics must be given. If the patient's sepsis does not respond to antibiotics, amputation must be considered (O'Malley et al 1993).

Thrombosis prophylaxis and pain relief

Thrombosis prophylaxis can be achieved with acetylsalicylic acid (aspirin), 300 mg/day for five days. Heparin has been tried but has not been shown to be beneficial. Administration of dextran 40 improves the results obtained in cases of extremity injury and is suggested at a drip rate of 25 ml/hour (O'Malley et al 1993). Rewarming can be very painful and opiates may be necessary for relief. Afterwards, aspirin or a non-steroidal anti-inflammatory medication will often suffice. Epidural pain relief can

be useful, both for immediate treatment of the pain and for achieving a sympathetic block (Taylor 1999) to improve the microcirculation. Stellate ganglion block for the upper extremity can have the same positive microcirculatory effect.

Hyperbaric oxygen has been used but evidence that it is beneficial is hard to find. If used, it should be administered during the first three days after injury. There is no evidence that later treatment helps (Okuboye and Ferguson 1968).

Long-term treatment

The treatment of a severe case takes many months. Physiotherapy, rehabilitation, reconstructive surgery and psychological help are all necessary. Pain on exposure to cold may persist for many years and be very difficult to treat.

Long-term sequelae

Pain or numbness on exposure to cold are the most frequent sequelae and may last a life-time. Other long-term complications include – hyperhidrosis, abnormal nail growth from fungal infections, arthritic changes (especially in children frozen before the epiphyses have fused), scaling of the skin, Raynaud's phenomenon, increased sensitivity to cold and an increased susceptibility to later cold injury. Many years after injury the victim might be found to employ a wide shuffling gait with most weight on the heels, due to pain and loss of proprioception.

Non freezing cold injury – NFCI – “trench foot” – “immersion foot”

Clinical picture

NFCI occurs after exposure of a limb, almost always the leg, to a moist, cool, environment for 2–3 days. The first sign is a numb, swollen, oedematous foot. Within a few days the leg may become very painful and so sensitive that the victim cannot walk. The initial colour is often red, but this soon pales and often becomes cyanotic. Pulses are difficult or impossible to feel because of the swelling. Left untreated, the swelling persists for six to eight weeks. This condition is often accompanied by aching, increased sensitivity to pain, fissures and infections. At a later stage, months or years after the injury, greater sensitivity to cold, Raynaud’s phenomenon, and swelling may occur.

Pathophysiology

The severity of the lesion depends on the temperature of the environment and the duration of exposure. The characteristic lesion is waterlogged stratum corneum of the skin, especially of the sole of the feet (Wrenn 1991). The superficial nerves may be damaged (Irwin 1997) as may the muscles and blood vessels in severe cases. There are no ice crystals in the tissue. During the 1940s Blackwood and Russel (1946; 1944) were able to demonstrate injuries following cold-wet exposure which affected all tissues, but above all the muscles and nerves. The picture included degenerative changes in nerve and muscle tissue and blood vessel walls.

A similar injury with waterlogged skin can occur with prolonged immersion in warm water (Humphrey and Ellyson 1997).

Treatment of non-freezing cold injuries

The treatment of NFCI is controversial, because no definitive treatment methods have had any impact on tissue survivability. Initial treatment includes careful washing and drying and elevation to diminish swelling. “Keep the body warm but the foot cool.” This reduces the metabolic requirements, while the body supplies oxygen and nutrition (Hamlet 1986) suggests the following:

1. Dry the foot well. Keep the body warm but the foot cool.
2. Put the patient to bed, with the foot raised. Keep the foot dry and cool with a fan.
3. Medicate for pain and infection as necessary.
4. Epidural sympathetic block with an opiate may be useful for control of pain early in treatment. Dextran 40 and heparin may be helpful to maintain blood flow.
5. Optimise nutrition with a high-protein, high calorie diet.
6. Avoid early surgery, but if C-reactive protein (CRP) rises and high fever or other indications of sepsis appear, early amputation must be considered.

Combined injuries

Non-freezing cold injuries and frostbite can occur simultaneously. Combinations of cold injury with fractures and lacerations are not uncommon, e.g. in connection with avalanche accidents. Lastly there are injuries caused by frostbite, spontaneous thawing and refreezing (refreezing injuries). These combined injuries are more difficult to treat than simpler injuries, and the end result is often poor.

Summary of recommendations for the care of hypothermic patients

Pre-hospital care

Do not pronounce death until certain

1. When taking care of a hypothermic person, assess:
   A – Airway
   B – Breathing
   C – Circulation
   D – Disability
   E – Exposure and environmental control, degrees and dry

Airway should be checked to ensure it is open and clear and that no danger of obstruction exists. If the airway is compromised, it will have to be opened using manual methods (chin lift or jaw thrust), advancing to mechanical means (oral, nasal airways, or endotracheal intubation). The indication for intubation is the same as in normothermic patients. Intubating the hypothermic patient may however, be more difficult, owing to stiff jaws and a proneness to mucosal bleeding. However, intubation must not be withheld by these considerations when the airway is threatened. Administer oxygen, 4–6 l/min on a bridle or 40–60 % with a mask, preferably warm and moisturised.

Cervical spine control – when establishing an open airway, there is always the possibility that a cervical spine injury may exist. Thus early immobilisation is essential in most severely injured patients.

Breathing-Ventilation – Check spontaneity of ventilation. If it is not spontaneous, ventilation is administered.

Circulation – circulatory system failure is just as life-threatening as failure of the respiratory system. Evaluate the pulse for presence, quality and regularity. All pulses in the hypothermic may be extremely difficult to find. First feel for the radial pulse, if impalpable feel for the femoral artery and as a last resort feel for a single carotid artery.

A major hazard in the hypothermic is life-threatening ventricular arrhythmias, cardiac arrest, although this is difficult to diagnose without an EKG.

CPR is not to be started until cardiac arrest has been proved. Remember that CPR has no effect on survival in the following situations and therefore is not to be started when:

(a) a drowned person has been under the water for more than an hour
(b) there are obvious fatal injuries
(c) the victim is frozen, with snow and ice in the airway
(d) the chest is so rigid that cardiac compression is impossible.

Disability – Determine the patient's level of consciousness (LOC). The patient's LOC can be accurately described by assigning one letter of the simple acronym AVPU that stands for

A – Alert
V – Responds to verbal stimulus
P – Responds to painful stimulus
U – Unresponsive

As soon as possible make a more thorough neurological examination using the Glasgow Coma Scale (GCS).

Exposure and environmental control, Degrees
Dry – Treat the patient to avoid further heat loss. Remove wet clothing **with extreme care,** insulate with blankets, sleeping bags, bubble-wrap, and/or rescue bags. Add extra **insulation to the head and feet.**

Degrees – Assess the temperature. Use a low-reading thermometer able to measure down to at least +25 °C. Measure the temperature orally, rectally or with a Metraux thermometer in the ear.

2. If the patient is conscious, administer a warm drink. If consciousness is depressed, administer fluids intravenously. Start with glucose 5 % in normal saline at a temperature of 37–40 °C, but do not delay evacuation by spending a lot of time on intravenous access. Consider intraosseous infusion if the establishment of intravenous access is difficult. In an unconscious patient, administer glucose 30 % 30 ml iv. and Naloxon 0.2–0.4 mg i.v. to prevent hypoglycaemia or opiate-induced coma.
3. Evacuate the patient as soon as possible to a facility where rewarming can take place under qualified supervision. Patients who *cannot* be evacuated are to be put in a room where the temperature is between 20 and 25 °C. Insulate the patient from further heat loss. If the patient is conscious and shivering, additional warmth may not be necessary. Continue to provide warm fluids with calories. If the patient is unconscious and not shivering, apply whatever warmth is available, taking care not to burn the skin with hot bottles, etc. The additional warmth will prevent further heat loss and may diminish after-drop.

4. Transport time is critical. The longer the patient remains cold, the greater the chance of complications and difficulty in management. Transportation must be gentle. Maintain the temperature in the ambulance or helicopter at a level that is comfortable for the rescuers, 25–30 °C.

- **WARNING:** Avoid direct application to the skin of hot objects such as hot water jugs. Do not rewarm in front of an open fire. If frostbite is present avoid the thaw-refreeze cycle.

Hospital care

Diagnosis and initial actions

Assess Airway, Breathing, Circulation, Disability, Exposure and environmental control, think Degrees and Dry

Airway and Breathing. The airway should be checked to ensure it is open and clear and that no danger of obstruction exists. If the airway is compromised, it will have to be opened using manual methods (chin lift or jaw thrust), advancing to mechanical means (oral, nasal airways, or endotracheal intubation). Cervical spine control – when establishing an open airway, there is always the possibility that a cervical spine injury may exist. Thus early immobilisation is essential

Consider the need for respiratory assistance. Administer oxygen, 4–6 l/min on a bridle or 40–60 % with a mask, preferably warm and moisturised. Connect a pulse oximeter. Insert a nasogastric tube for gastric drainage.

Circulation – Consider the need for, haemostasis, shock prevention and CPR. Monitor the EKG with defibrillation backup. If CPR is indicated, start at half to normal speed. Treat life-threatening arrhythmias. Insert intravenous lines, a central venous line and an arterial catheter. Measure arterial pressure and CVP. Correct dehydration, and restore normal blood volume. Insert an indwelling bladder catheter, measure hourly urinary output. If necessary stimulate adequate diuresis – primarily using mannitol.

Disability – Determine the patient's level of consciousness (LOC) using the Glasgow Coma Scale (GCS). As soon as possible make a more thorough neurological examination.

Exposure and environmental control, degrees

- Remove wet clothing with extreme care.
- Carry out a whole body examination.
- Monitor body temperature. The most accurate site is the mid-oesophagus. If this is not possible, rely on rectal or bladder temperature. Tym-

panic temperatures may not be accurate and oral and axillary temperature readings are almost certain to be inaccurate compared with a true core temperature.

Laboratory tests
Laboratory tests must include: blood-glucose, blood gas, blood count, electrolytes, liver function tests, cardiac markers, including CK, CKMB, and Troponin-I or Troponin-T, coagulation status, urea, magnesium, and amylase. Consider a toxic screen if there is any suspicion of drug ingestion.

X-ray the lungs, cervical spine and any other appropriate area of an unconscious patient who has been subjected to trauma.

Rewarming

The choice of rewarming method will depend on the patient's condition, the cause of the hypothermia, and the resources and knowledge available.

Guidelines:

Body temperature >32 °C
- Passive rewarming. Insulate with warm, dry blankets.
- Room temperature 25 °C.
- Supply a conscious patient with warm drink and calories.
- Supply an unconscious patient with warm infusion solutions, start with glucos 5% in normal saline, 37–40 °C.

Body temperature 32–28 °C
- The choice of rewarming method will depend on the degree of consciousness and the available resources.
  (a) A conscious patient can be rewarmed passively, but if there are local cold injuries choose an active method if the resources and knowledge for active rewarming are available.
  (b) A partially conscious or unconscious patient must be actively rewarmed if optimum resources (see below) are available. Failing this, the rule is passive rewarming.

Body temperature <28 °C
Active rewarming must be performed if optimum resources are available. Failing this, rewarm passively

Optimum resources comprise:
An intensive care unit with facilities for close monitoring and preparedness for the treatment of arrhythmias, fluid balance and electrolytic dis-

turbances, equipment for rapid rewarming, knowledge of the changes expected in a hypothermic patient during rewarming.

The following rewarming methods can be considered:

External rewarming	Internal rewarming
Spontaneous rewarming	Warm air inhalation (40–45 °C)
Warm blankets, thermal ceiling	Peritoneal lavage (40–45 °C)
Forced air warming	Pleural lavage (40–42 °C)
Bath tub (40–42 °C)	Haemodialysis
	Extracorporeal circulation

Generally speaking, a patient with *chronic hypothermia*, which has developed slowly for a number of days, is metabolically worse off than somebody who has developed *acute hypothermia* in a matter of hours or minutes.

In the *chronically hypothermic* patient, glycogen stores have been exhausted, and there may have been heavy losses of fluid, both through cold-diuresis and a "fluid shift" to the extracellular space. Electrolyte disturbances have had time to develop, mainly with potassium. In the *acutely hypothermic* patient these changes are far less pronounced. When rewarming a chronically hypothermic patient, therefore, be prepared for greater disturbances in all physiological parameters, compared with those in an acutely hypothermic patient.

Whatever the method of rewarming adopted, extremely close monitoring is needed and preparedness to:

- Follow the acid-base balance.
- Follow the fluid balance.
- Follow and correct, if necessary, the potassium levels.
- Correct dehydration, and restore normal blood volume.
- Stimulate adequate diuresis – primarily using mannitol.
  Treat life-threatening arrhythmias.

Conveyance to more advanced care

This may be necessary in cases of:

- Inadequate local resources.
- Profound unconsciousness.
- Hypothermia combined with extensive trauma requiring surgery.
- Hypothermia combined with severe frostbite.

Before being transferred, the patient must be stabilised and the receiving hospital amply prepared.

Summary

The guiding principles of actions must be:

- Do not prematurely pronounce a patient dead.
- Be gentle.
- Protect and shelter the victim.
- Insulate from further heat loss.
- Transfer gently to a rewarming facility.
- Do not hesitate to seek advice.

Disasters – Priority guidelines

In situations involving large numbers of injured people, very difficult problems of prioritisation, triage, are liable to occur because many patients will need a lot of care at the same time.

Triage is the process of sorting patients into priorities in order to establish an order for treatment and evacuation. Triage takes many different forms, and operates at a number of different levels, but its overall aim at all times is to provide the right patient with the right care at the right time in the right place. Triage must be a simple procedure that is swift, but reliable and reproducible.

A common pre-hospital Priority "P" system in use in Sweden is summarised in the table below.

Description	P	Colour
Immediate	1	Red
Urgent	2	Yellow
Delayed	3	Green
Dead		Black/white

Immediate priority:	Red casualties requiring immediate procedures to save life e.g. airway obstruction, tension pneumothorax.
Urgent priority:	Yellow casualties requiring medical treatment within 4–6 hours e.g. compound fractures.
Delayed priority:	Green casualties with injuries that can wait for treatment until after 4–6 hours, or casualties with non-survivable injuries.
Dead:	Black/white dead casualties must be identified and clearly labelled as such to avoid re-triage.

A fast, simple and reliable assessment of the patient at the scene is necessary. The "Triage Sieve" is a system used in UK, based on mobility, followed by a simple assessment of ABC. The capillary refill time (CRT) gives an indication of peripheral perfusion and is simple and rapid to ascertain. Environmental conditions, especially dark and cold make the use of CRT difficult. If this is the case the pulse should be used. See table below**.**

Table 3. The Triange Sieve

Mobility	Can the patient walk?	yes		→	*P3 Delayed*
		no		→	assess A and B
Airway and breathing	Is the patient breathing?	no	→	open airway, breathing now?	
			no	→	*Dead*
			yes	→	*P1 Immediate*
		yes	→	assess rate	
			<10 or >29	→	*P1 Immediate*
			10–29	→	assess C
Circulation	CRT*	>2 s			
		or pulse >120 beats/min		→	*P1 Immediate*
	CRT	<2 s			
		or pulse <120 beats/min		→	*P2 Urgent*

* CRT=Capillary refill time.

The simple triage and rapid system (START) is an alternative system in use in the United States. Table below.

Table 4. Thesimple triage and rapid treatment system (START)

Is the patient breathing?	no	→	open airway, breathing now?	
		no	→	*Dead*
		yes	→	*Immediate care*
	yes	→	assess rate	
			>30	→ *Immediate care*
			<30	→ check radial pulse
Radial pulse present?		no	→	control haemorrhage
			→	*Immediate care*
		yes	→	assess mental state
Following commands		no	→	*Immediate care*
		yes	→	*Urgent care*

However there are considerations in dealing with hypothermic victims that are not the same as when dealing with normothermic victims. Whichever system is used for triage, the CRT, pulse and breathing may be very difficult to assess in a hypothermic victim and so is not immediately applicable in the hypothermic situation.

The rule about putting the apparently dead in the last category for aid and transportation does not apply in the hypothermic victim.

If the disaster is predominantly one involving cold e.g. several people rescued from a capsized boat in an icy river, assess the temperature of the victims. If deep hypothermia is a possibility and a victim appears to be dead and does not have any obviously fatal injuries, this person should be transported in the first group, that is with the highest priority.

In the hypothermic situation **casualties are to be regarded as living until rewarming and monitoring have taken place in hospital (see hospital care), unless they satisfy the following criteria for pronouncement of death:**

- A drowned person who has been under the water for more than an hour.
- Obvious fatal injuries.
- Packed snow or ice in the respiratory tract.

Priority guidelines in the hypothermic situation

Prioritisation for transportation to a medical facility is affected by the ability to measure a dependable body temperature.

Impossible to measure body temperature

High priority ("very urgent") is given to *suspected hypothermic patients* with:

- impaired vital functions
- reduced consciousness or unconsciousness
- profound hypothermia and apparently dead
- simultaneous severe trauma.

Low priority ("non-urgent") is given to *suspected hypothermia patients* with:

- vital functions unaffected
- no simultaneous trauma
- no signs of life, apparent mild hypothermia, reason to believe suffocation preceded cooling (submersion >15 minutes, buried in avalanche for >30 minutes).

If a thermometer is available and the situation permits temperature measurement, priorities are as follows

High priority ("very urgent") is given to patients:

- with vital functions impaired
- with body temperatures below 28 °C and/or reduced consciousness,
- with significant trauma and temperatures below 35 °C.

Medium priority ("urgent") is given to patients with:

- vital functions unaffected
- body temperatures 28–32 °C but no reduction of consciousness
- body temperature 28–32 °C but with no simultaneous trauma.

Low priority ("non-urgent") is given to patients with:
- temperature 32–35 °C and vital functions normal
- body temperature 32–35 °C and no reduction of consciousness
- no simultaneous trauma
- no signs of life and meeting the criteria for death.

Hospital care

In many major disasters, available resources are immediately outstripped by needs. The treatment of hypothermic patients must conform to the guidelines for situations without optimal resources, i.e. passive rewarming.

If resources for optimum care of individual patients can be released without other patients being made to suffer, handling should of course, be modified accordingly.

References

Albiin N, Eriksson A: Fatal accidental hypothermia and alcohol. Alcohol Alcoholism 19:13, 1984.

Althaus U et al. Management of profound accidental hypothermia with cardiorespiratory arrest. Ann Surg 195:492, 1982.

Altus P, Hickman JW, Nord HJ. Accidental hypothermia in a healthy quadriplegic patient. Neurology 35:427, 1985.

Andrews RP. Cold injury complicating trauma in the subfreezing environment. Milit Med 152:42, 1987.

Angelakos ET. Influence of pharmacologicalal agents on spontaneous and surgically-induced hypothermic ventricular fibrillation. Ann NY Acad Sci 80:351, 1959.

Ariev TJ. Monograph on frostbite. Narkomzdrav, USSR, State Health Committee: 1940. Translated by Defence Research Board, Canada, 1955.

Ashworth M et al. Missed injuries of the spinal cord. Br Med J 284:1334, 1982.

Astrup J et al. Increase in extracellular potassium in the brain during circulatory arrest: effects of hypothermia, lidocaine, and thiopental. Anesthesiology 55:256, 1981.

Auerbach PS. Some people are dead when they're cold and dead (editorial). JAMA 264:1856, 1990.

Bangs CC, Hamlet MP. Hypothermia and cold injuries. In Auerbach P. Geehr E, editors: Management of wilderness and environmental emergencies. New York, 1983, Macmillan.

Bangs CC. Hypothermia and frostbite. Emerg Med Clin North Am 2:275, 1984.

Baraka A. Hydrogen ion regulation during hypothermia: hibernators versus ectotherms. Middle East J Anesth 7:235, 1984.

Baraka AS et al. Effect of alpha-stat versus pH-stat strategy on oxyhemoglobin dissociation and whole-body oxygen consumption during hypothermic cardiopulmonary bypass. Anesth Analg 74:32, 1992.

Barker JR et al. Magnetic resonance imaging of severe frostbite injuries. Ann Plast Surg 38:275, 1997.

Beyda EJ, Jung M, Bellet S. Effect of hypothermia on the tolerance of dogs to digitalis. Circ Res 9:129, 1961.

Bierens JJ et al. Accidental hypothermia: incidence, risk factors and clinical course of patients admitted to hospital. Eur J Emerg Med 2:38, 1995.

Biggar WD, Bohn D, Kent G. Neutrophil circulation and release from bone marrow during hypothermia. Infect Immun 40:708, 1983.
Bjornstad H, Tande PM, Refsum H. Cardiac electrophysiology during hypothermia: implications for medical treatment. Arctic Med Res 50:71, 1991.
Blackwood W, Russel H. Experiments in the study of immersion foot. Edinb Med J 50 No 7, July 1943.
Blackwood W. Studies in the pathology of human immersion foot. Br J 31:329, 1944.
Blair E. Physiology of hypothermia. In Clinical Hypothermia. New York, 1964, McGraw-Hill.
Boelhouwder RU, Bruining HA, Ong GL. Corrections of serum potassium fluctuations with body temperature after major surgery. Crit Care Med 15:310, 1987.
Bolgiano E et al. Accidental hypothermia with cardiac arrest: recovery following rewarming by cardiopulmonary bypass. J Emerg Med 10:427, 1992.
Bolte RG et al. The use of extracorpareal rewarming in a child submergerd for 66 minutes. JAMA 5:377, 1998.
Boutelier C. Survival and protection of aircrew in the event of accidental immersion in cold water. AGARD Report No AG-211, Neuilly Sur Seine, France: NATO 1979.
Bowman W. Outdoor emergency care: comprehensive first aid for non-urban settings. Lakewood. CO, 2nd Ed. 1993, National Ski Patrol System.
Brantigan CO, Paton B: Clinical hypothermia, accidental hypothermia and frostbite. In Goldsmith HS, editor: Lewis practice of surgery, New York, Harper and Row, 1978.
Brauer A, et al. Severe accidental hypothermia: rewarming strategy using a veno-venous bypass system and a convective air warmer. Intensive Care Med 25:520, 1999.
Brinnel H, Cabanac M. Tympanic temperature is a core temperature in humans. J Therm Biol 14:47, 1989.
Bristow G. Treatment of accidental hypothermia with peritoneal dialysis. Can Med Assoc J 118:764, 1978.
Britt LD, Dascombe WH, Rodriguez A. New horizons in management of hypothermia and frostbite injury. Surg Clin North Am 71:345, 1991.
Brunette DD et al. Comparison of gastric and closed thoracic cavity lavage in the treatment of severe hypothermia in dogs. Ann. Emerg. Med. 16:1222, 1987.
Brunette DD et al. Internal cardiac massage and mediastinal irrigation in hypothermic cardiac arrest. Am J Emerg Med 10:32, 1992.

Budd GM. Accidental hypothermia in skiers (editorial). Med J Aust 144:449,1986.
Buky B. Effect of magnesium on ventricular fibrillation due to hypothermia. Br J Anaesth 42:886, 1970.
Caldwell C, Crawford R, Sinclair I. Hypothermia after cardiopulmonary bypass in man. Anesthesiology 55:86, 1981.
Candler WH, Ivey H. Cold weather injuries among US soldiers in Alaska: a five-year review. Mil Med 162:788, 1997.
Carden DL, Nowak RM. Disseminated intravascular coagulation in hypothermia. JAMA 247:2099, 1982.
Carden DL. Intubating the hypothermic patient. Ann Energ Med 12:124,1983.
Catermole TJ. The epidemiology of cold injury in Antarctica. Aviat Space Environ Med 70:135, 1999.
Chang M, Gill T. Hypothermia, neurological dysfunction, and sudden death in a man with carcinoma. South Med J 74:1509, 1981.
Chochinov AH et al. Recovery of a 62-year old man from prolonged cold water submersion. Ann Emerg Med 31:127, 1998.
Clardy CW, Edwards KM, Gay JC. Increased susceptibility to infection in hypothermic children: possible role of acquired neutrophil dysfunction. Pediatr Infect Dis 4:379, 1985.
Clayton DG et al. A comparison of the performance of 20 pulse oximeters under conditions of poor perfusion. Anaesthesia 46:3, 1991.
Clements SD, Hurst JW. Diagnostic value of electrocardiographic abnormalities observed in subjects accidentally exposed to cold. Am J Cardiol 29:729, 1972.
Cohen DN. Rewarming with hot packs. Am J Emerg Med 3:371, 1985.
Conway GA, Husberg BJ. Cold–related non-fatal injuries in Alaska. Am J Ind Med 1:39-41, 1999.
Covino BG, Beavers WR. Changes in cardiac contractility during immersion hypothermia. Am J Physiol 195:433, 1958.
Crino MH, Nagel EL. Thermal burns caused by warming blankets in the operating room. Anesthesiology 29:149, 1968.
Dagan R, Gorodischer R. Infections in hypothermic infants younger than 3 month old. Am J Dis Child 183:483, 1984.
Dahlgren BE, Nilsson HG, Viklund B. Tracheal tubes in cold stress. Anaesthesia 43:683, 1988.
Danielsson U. Windchill and the risk of tissue freezing. J Appl Physiol 81:2666-73, 1996.
Danzl DF, Thomas DM. Nasotracheal intubations in the emergency department. Crit Care Med 8:677, 1980.
Danzl DF et al. Chemical ventricular defibrillation in severe accidental hypothermia. Ann Emerg Med 11:698, 1982.

Danzl DF. Accidental hypothermia. In Rosen et al, editors: Concepts and clinical practice. New York, 1983, Mosby.
Danzl DF et al. Multicenter hypothermia survey. Ann Emerg Med 16:1042, 1987.
Danzl DF. Accidental hypothermia. In Rosen et al, editors: Emergency medicine: concepts and clinical practice, 2nd Ed., St Louis, 1988, Mosby.
Danzl DF et al. Hypothermia outcome score: development and implications. Crit Care Med 17:227, 1989.
Danzl DF. Blood flow during closed chest compression in hypothermic humans. Wilderness Med 7:12, 1991.
Danzl DF, Pozo RS, Hamlet MP. Accidental hypothermia. In Auerbach PS, Geehr EC editors: Management of wilderness and environmental emergencies, ed 2, St Louis, 3rd Ed.1998, Mosby.
Danzl DF.Accidental Hypothermia, In Auerbach PS, editor: Wilderness Medicine 4th edition, 2001,St Louis, Mosby.
Darowski A et al. Hypothermia and infection in elderly patients admitted to hospital. Age Ageing 20:100, 1991.
DaVee TS, Reineberg EJ. Extreme hypothermia and ventricular fibrillation. Ann Emerg Med 9:100, 1980.
Davies DM, Millar EJ, Miller IA. Accidental hypothermia treated by extracorporeal blood-warming. Lancet 1:1036, 1967.
Deakin CD. Forced air surface rewarming in patients with severe accidental hypothermia. Resuscitation 43:223, 2000.
Deal CW, Warden JC, Monk I. Effects of hypothermia on lung compliance. Thorax 25:105, 1970.
Deimi R, Hess W. Successful therapy of a cardiac arrest during accidental hypothermia using extracorporeal circulation. Anaesthesist 41:93, 1992.
Desmeules H, Blais C. Hypothermie accidentelle: traitement d'un cas par irrigation peritoneale (Accidental hypothermia: treatment of a case using peritoneal irrigation). Can Anaesth Soc J 26:506, 1979.
Diaz JH, Cooper ES, Ochsner JL. Cold hemaglutination pathophysiology. Arch Intern Med 144:1639, 1984.
Dorsey JS. Venoarterial bypass in hypothermia. JAMA 244:1900, 1980.
Duguid H, Simpson RG, Stowers JM. Accidental hypothermia. Lancet 2:1213, 1961.
Dundee JW, Clarke RSJ. Pharmacology of hypothermia. Int Anesth Clin 2:857, 1964.
Edelman IS. Thyroid thermogenesis. N. Engl J Med 290:1303, 1974.
Edwards HA et al. Apparent death with accidental hypothermia. Br J Anesth 42:906, 1970.

Edwards RJ, Belyavin AM, Harrison MH: Core temperature measurement in man. Aviat Space Environ Med 49:1289, 1978.
Ehrenkranz JRL. Urine temperature and core temperature. JAMA 255:1880, 1986.
Ehrmantraut WR, Ticktin HE, Fazekas JF. Cerebral hemodynamics and metabolism in accidental hypothermia. Arch Intern Med 99:57, 1957.
Eisenberg PR, Jaffe AS, Schuster DP. Clinical evaluation compared to pulmonary artery catheterization in the hemodynamic assessment of critically ill patients. Crit Care Med 12:549, 1984.
Emslie-Smith D. Accidental hypothermia: a common condition with a pathognomonic electrocardiogram. Lancet 2:492, 1958.
Ervasti O et al. Frostbite in reindeer herders. Arct Med Res 50: Suppl 6,89093,1991.
Falk RH, Zoll PM, Soll RH. Safety and efficacy of noninvasive cardiac pacing: a preliminary report. N Engl J Med 309:1166, 1983.
Faller JP, Rauscher M. Severe hypothermia: importance of temporary electrosystolic pacing until the end of the warming. Nouv Presse Med 7:3366, 1978.
Feldman KW, Morray JP, Schaller RT. Thermal injury caused by hot pack application in hypothermic children. Am J Emerg Med 3:38, 1985.
Ferguson NV. Urban hypothermia. Anesthesia 40:651, 1985.
Fernandez JP, O'Rouke RA, Ewy GA. Rapid active external rewarming in accidental hypothermia. JAMA 212:153, 1970.
Ferrara et al. Hypothermia and acidosis worsens coagulopathy in the patient requiring massive transfusion. Am J Surg 160:515, 1990.
Ferraro FJ Jr et al. Cold-induced hypocoagulability in vitro, a trauma connection? Am Surg 58:355, 1992.
FitzGibbon FT, Hayward JS, Walker D. EEG and visual evoked potentials of conscious man during moderate hypothermia. Electroencephalogr Clin Neurophysiol 58:48, 1984.
Foray J. Mountain frostbite. Current trends in prognosis and treatment (from the results concerning 1261 cases) Int J Sports Med 13: S193, 1992.
Fox RH et al. Hypothermia in a young man with an anterior hypothalamic lesion. Lancet 2:185,1970.
Francis TJR. Non-freezing cold injury: a historical review, J R Nav Med Serv 70:134, 1984.
Frank DH, Robson MC. Accidental hypothermia treated without mortality. Surg Gynecol Obstet 151:379, 1980.
Froese G, Burton AC, Heat loss from the human head. J Appl Physiol 10: 235, 1957.

Fuhrman FA, Crismon JM. Studies on gangrene following cold injury. VII. Treatment of cold injury by means of immediate rapid rewarming. J Clin Invest. 26:476, 1947.
Gentilello LM et al. Continuous arteriovenous rewarming: rapid reversal of hypothermia in critically ill patients. J Trauma 32:316, 1992.
Gentilello LM, Rifley WJ. Continuous arteriovenous rewarming: report of a new technique for treating hypothermia. J Trauma 31:1151, 1991.
George A, et al. Liquid ammonia injury. Burns 26:409, 2000.
Giesbrecht GG, and GK Bristow. A second post-cooling afterdrop: evidence for a convective mechanism. J Appl Physiol, 32:316, 1992
Giesbrecht GG et al. Treatment of mild immersion hypothermia by direct body-to-body contact. J Appl Physiol 76:2373, 1994.
Giesbrecht GG, Pachu P, Xu X. Design and evaluation of a portable rigid forced-air warming cover for prehospital transport of cold patients. Aviat. Space and Environ. Med. 69:1200, 1998.
Giesbrecht GG, et al. Immersion of distal arms and legs in warm water effectively rewarms mildly hypothermic humans. J Wilderness and Environ Med 11:59, 2000.
Giesbrecht GG. Cold stress, near drowning and accidental hypothermia: a review. Aviat Space and Environ Med.71:733, 2000.
Gilbert M et al. Resuscitation from accidental hypothermia of 13.7 degrees C with circulatory arrest. Lancet 29:375, 2000.
Gillen JP et al. Ventricular fibrillation during orotracheal intubation of hypothermic dogs. Ann Emerg Med 15:412, 1986.
Glick SD, Guido RA. Naloxone antagonism of the thermoregulatory effects of phencyclidine. Science 217:1272, 1982.
Glusman A, Hasan K, Roguin N. Contraindication to thrombolytic therapy in accidental hypothermia. Int J Cardiol 28:269, 1990.
Goheen S et al. Efficacy of forced-air and inhalation rewarming using a human model for severe hypothermia. J Appl Physiol. 83:1635, 1997.
Golden FStC, Hervey GR. The mechanism of the after-drop following immersion hypothermia in pigs. J Physiol 272:26P, 1977.
Goldman et al. A pilot study of low body temperature in old people admitted to hospital, J R Coll Phys Lond 11:291, 1977.
Granberg PO. Alcohol and cold. Arctic Med Res 50:43, 1991.
Granberg PO. Human physiology under cold exposure. Arctic Med Res. 50:23, 1991.
Gravlee GP. Heparin-coated cardiopulmonary bypass circuits. J Cardiothorac Vasc Anesth Apr: 8(2):213, 1994.
Greenwald D et al. An algorithm for early aggressive treatment of frostbite with limb salvage directed by triple-phase scanning. Plast and Reconstruct Surg 102:1069, 1998.

Gregory JS et al. Comparison of three methods of rewarming from hypothermia; advances of extracorporeal blood rewarming. J Trauma 31:1247, 1991.
Gregory RT et al. Treatment after exposure to cold. Lancet 1:377, 1972.
Grice KA, Bettley FR. Skin water loss and accidental hypothermia in psoriasis, ichthyosis, and erythroderma. Br Med J 4:195, 1957.
Grossheim RL. Hypothermia and frostbite treated with peritoneal dialysis. Alaska Med 15:53, 1973.
Guerin JM, Meyer P, Segrestaa JM. Hypothermia in diabetic ketoacidosis. Diabetes Care 10:801, 1987.
Hall KN. Closed thoracic cavity lavage in the treatment of severe hypothermia in human beings. Ann Emerg Med 19:204, 1990.
Hamlet MP. The fluid shifts in hypothermia. In Pozos RS, Wittmers LE, editors: The nature and treatment of hypothermia, Minneapolis, 1983, University of Minnesota Press.
Hamlet MP. Human cold injuries. In Pandolf KB, Sawka MN, Gonzalez RR, editors; Human performance physiology and environmental medicine at terrestrial extremes. Carmel IN, 1986, Cooper Publ. Group.
Harari A et al. Haemodynamic study of prolonged deep accidental hypothermia. Eur J Intensive Care Med 1:65, 1975.
Harnett RM et al. Initial treatment of profound accidental hypothermia. Aviat Space Environ Med 51:680, 1980.
Harnett RM, Pruitt JR, Sias FR. A review of the literature concerning resuscitation from hypothermia. I. The problem and general approaches. Aviat Space Environ Med 54:425, 1983.
Harnett RM, Pruitt JR, Sias FR. A review of the literature concerning resuscitation from hypothermia. II. Selected rewarming protocols. Aviat Space Environ Med 54:487, 1983.
Hashmi MA, et al. Frostbite: epidemiology at high altitude in the Karakoram mountains. Ann R Coll Surg Engl 80:91, 1998.
Hauty MG et al. Prognostic factors in severe accidental hypothermia: experience from the Mt. Hood tragedy. J Trauma 27:1107,1987.
Hayward J, Eckerson J. Physiological responses and survival time prediction for humans in ice-water. Aviat Space Environ Med 55:206, 1984.
Hayward JS, Eckerson JD, Collis ML. Thermal balance and survival time prediction of man in cold water. Can J Physiol Pharmacol 53:21, 1975.(a)
Hayward JS, Eckerson J, Collis ML. Effect of behavioral variables on cooling rate of man in cold water. J Appl Physiol 38:1073, 1975.(b)
Hayward JS, Steinman AM. Accidental hypothermia: an experimental study of inhalation rewarming. Aviat Space Environ Med 46:1236, 1975.

Hayward MG, Keatinge WR. Roles of subcutaneous fat and thermoregulatory reflexes in determining ability to stabilize body temperature in water. J Physiol London 320:229, 1981.
Heggers JP et al. Experimental and clinical observations on frostbite. Ann Emerg Med 169:1056, 1987.
Hernandez E et al. Hemodialysis for treatment of accidental hypothermia. Nephron 63:214, 1993.
Himms-Hagen J. Thermogenesis in brown adipose tissue as an energy buffer. N Engl J Med 311:1549, 1984.
Hirvonen J, Huttunen P. Necropsy findings in fatal hypothermia cases. Forensic Sci 8:155,1976.
Holoday JV, Fadel AI. Naloxone acts at central opiate receptors to reverse hypotension, hypothermia, and hypoventilation in spinal shock. Brain Res 189:295,1980.
Hoskin RW, Melinskyn MJ, Romet TT, Goode RC. Bath rewarming from immersion hypothermia. J Appl Phys 61(4) 1515, 1986.
Humphrey W, Ellyson R. Warm water immersion foot: still a threat to the soldier. Mil Med 162:610, 1997.
Husby P et al. Accidental hypothermia with cardiac arrest: complete recovery after prolonged resuscitation and rewarming by extracorporeal circulation. Intensive Care Med 16:69, 1990.
Husby P et al. Deep accidental hypothermia treated with heart-lung machine after prolonged cardiopulmonary resuscitation. Tidsskr Nor Laegeforen 111:183, 1991.
Huttunen P, Hirvonen J. The effect of ethanol on the ability of guinea pigs to survive severe cold. Forensic Science 9, 185, 1977.
Irone M et al. Extracorporeal membrane oxygenation in emergency resuscitation from deep hypothermia. Perfusion 13:211, 1998.
Irwin MS et al. Neuropathy in non-freezing cold injury (trench foot) J Roy Soc Med 90:433, 1997.
Iyengar J, Bhakoo ON. Prevention of neonatal hypothermia in Himalayan village: role of domiciliary caretaker. Trop Geogr Med 43:293, 1991.
Jessen K, Hagelsten JO. Peritoneal dialysis in the treatment of profound accidental hypothermia. Aviat Space Environ Med 49:426, 1978.
Jessen K. Immersion and accidental hypothermia. Acta Med Port 2:225, 1979.
Johnson LA. Accidental hypothermia: peritoneal dialysis. JACEP 6:556, 1977.
Johnston CE, Bristow GK, Elias D, Giesbrecht GG. Alcohol lowers the vasoconstriction threshold in humans without affecting core cooling rate during mild exposure. Eur J Appl Physiol. 74: 293, 1996

Johnston TD, Chen Y, Reed R, 2nd. Functional equivalence of hypothermia to specific clotting factor deficiences. J Trauma Sep:37(3):413, 1994.
Jones AI, Swann IJ. Prolonged resuscitation in accidental hypothermia: use of mechanical cardio-pulmonary resuscitation and partial cardiopulmonary bypass. Eur J Emerg Med 1:34, 1994.
Jonsson DE, Gamble WB. Trauma in the arctic: an incident report. J Trauma 31:1340, 1991.
Jurkovich GJ et al. Hypothermia in trauma victims: an ominous predictor of survival. J Trauma 27:1019, 1987.
Kallenback J et al. Experience with acute poisoning in an intensive care unit: a review of 103 cases. S Afr Med J 59:587, 1981.
Kangas AC et al. Treatment of hypothermic circulatory arrest with thoracotomy and pleural lavage. Ann Chir Gynaecol 83:258, 1994.
Kashuk JL et al. Major abdominal vascular trauma: a unified approach. J Trauma 22:672, 1982.
Keatinge WR. The effect of work and clothing on the maintenance of body temperature. Quart J Exp Physiol 46:69, 1961.
Keatinge WR. Survival in cold water. Oxford, U.K, Blackwell Scientific, 1969.
Keatinge WR et al. Increased platelet and red cell counts, blood viscosity, and arterial pressure during mild surface cooling: factors in mortality from coronary and cerebral thrombosis in winter. Br Med J 289:1405, 1984.
Kernbach-Wighton G et al. Clinical and morphologic aspects of death due to liquid nitrogen. Int J Legal Med 111:191, 1998.
Klarskov P, Amter F. Hypothermia after submersion: correction with peritoneal dialysis. Ugeskr Laeger 138:1937, 1976.
Knize D, Weatherly-White RCA, Paton B et al. Prognostic factors in the management of frostbite. J Trauma 9:749, 1969.
Kochar G, Kahn SE, Kotler MN. Bretylium tosylate and ventricular fibrillation in hypothermia. Ann Intern Med 105:624, 1986.
Koht A, Cane R, Cerullo LJ. Serum potassium levels during prolonged hypothermia. Intensive Care Med 9:275, 1983.
Kolodzik PW et al. The effects of antishock trouser inflation during hypothermic cardiovascular depression in the canine model. Am J Emerg Med 6:584,1988.
Kornberger E et al. Forced air surface rewarming in patients with severe accidental hypothermia. Resuscitation 41:105, 1999.
Krakauer J. Into Thin Air: a personal account of the Mount Everest disaster. Villard, New York. 1997.
Krook G. Hypothermia in patients with exfoliative dermatitis. Acta Derm Venereol 40:142, 1960.

Kugelberg J et al. Treatment of accidental hypothermia. Scand J Thorac Cardiovasc Surg 1:142, 1967.
Kukarni RG, Thomas SH. Severe accidental hypothermia: the need for prolonged aggressive resuscitative efforts. Prehosp Emerg Care 3:254, 1999.
Kuspis DA, Krenezlok EP. Oral frostbite injury from the intentional abuse of a fluorinated hydrocarbon. J Toxicol Clin Toxicol 17:873, 1999.
Lamke LO, Liljedahl SO, Lennquist S. The influence of cold stress on cathecholamine excretion and oxygen uptake in normal persons. Scand J Clin Lab Invest 30:57, 1972.
Larrey, Baron DJ. Surgical memoirs of the campaigns of Russia, Germany and France. Translated from the French by JC Mercer. Philadelphia. Lea and Carey 1832:72.
Laub GW et al. Percutaneous cardiopulmonary bypass for the treatment of hypothermic circulatory collapse. Ann Thorac Surg 47:608, 1989.
Lennquist S. Sodium reabsorption in cold diuresis. Swedish Defence Med 7:108, 1971.
Lennquist S. Cold-induced diuresis, a study with special reference to electrolyte excretion, osmolal balance and hormonal changes. Scand J Urol and Nephrol. Suppl 9. 1972.
Lennquist S, Granberg P-O, Wedin B. Fluid balance and physical work capacity in humans exposed to cold. Arch Environ Health. 29:241, 1974.
Lewin S, Brettman LR, Holzman RS. Infections in hypothermic patients. Arch Intern Med 141:920, 1981.
Lewis T. Observations upon the reactions of the vessels of the human skin to cold. Heart 15:177, 1930.
Lexow K. Severe accidental hypothermia: survival after 6 hours 30 minutes of cardiopulmonary resuscitation. Arctic Med Res 50:112, 1991.
Lilly JK et al. Urinary bladder temperature monitoring: a new index of body core temperature. Crit Care Med 8:742, 1980.
Linko K, Honkavaara P, Nieminen MT. Heated humidification in major abdominal surgery. Eur J Anaesthesiol 1:285, 1984.
Livingston JH, Groggings RC. Clinical curio: hypothermia caused by treatment of a scald. Br Med J 228:771, 1984.
Lloyd EL, Mitchell B. Factors affecting the onset of ventricular fibrillation in hypothermia. Lancet 2:1294, 1974.
Lloyd EL. Equipment for airway warming in the treatment of accidental hypothermia. J Wilderness Med 2:330, 1991.
Lloyd EL. Accidental hypothermia. Resuscitation 32:111, 1996.

Locher T et al. Accidental hypothermia in Switzerland (1980-1987): case reports and prognostic factors. Schweiz Med Wochenschr 121:1020, 1991.
Mackowiak PA, Wasserman SS, Levine MM: A critical appraisal of 98,6°F, the upper limit of the normal body temperature, and other legacies of Carl Reinhold August Wunderlich. JAMA 268:1578, 1992.
Maclean D, Emslie-Smith D. Accidental hypothermia, Philadelphia, 1977, JB, Lippincott.
MacLean D, Murison J, Griffiths PD. Acute pancreatitis and diabetic ketoacidosis in hypothermia. Br Med J 2:59, 1974.
Mair P et al. Prognostic markers in patients with severe accidental hypothermia and cardiocirculatory arrest. Resuscitation 27:47, 1994.
Mair P et al. Successful resuscitation of a patient with severe accidental hypothermia and prolonged cardiocirculatory arrest using cardiopulmonary bypass. J Cardiothorac Vasc Anest 11:901, 1997.
Mazala M, Horrow JR, Storella RJ. Hypothermia and the action of neuromuscular blocking agents. Anaesthesia 43:162, 1988.
Mazur P. Cryobiology: The freezing of biological systems. Science 168:939, 1970.
McDonald JL. Coarse ventricular fibrillation presenting as asystole or very low amplitude ventricular fibrillation. Crit Care Med 10:790, 1982.
Mecklenburg RS et al. Hypothalamic dysfunction in patients with anorexia nervosa. Medicine 53:147, 1974.
Menard MR, Hahn G. Acute and chronic hypothermia in a man with spinal cord injury: environmental and pharmacological causes. Arch Phys Med Rehabil 74:421, 1991.
Meriwether WD, Goodman RM. Severe accidental hypothermia with survival after rapid rewarming. Am J Med 53:505, 1972.
Meryman HT. Mechanics of freezing in living cells and tissues. Science 1124:3221, 1956.
Meryman HT. Osmotic stress as a mechanism of freezing injury. Cryobiology 8:489, 1971.
Michenfelder JD, Milde JH. The relationship among canine brain temperature, metabolism, and function during hypothermia. Anesthesiology 75:130, 1991.
Miller JW, Danzl DF, Thomas DM. Urban accidental hypothermia: 135 cases. Ann Emerg Med 9:456, 1980.
Miller JW, Danzl DF, Thomas DM. Hypothermia treatment. Ann Emerg Med 10:396, 1981.
Mills WJ et al. Frostbite: experience with rapid rewarming and ultrasonic therapy. Wilderness and Environ Med. 9:226,1998.
Mills WJ. Frostbite: A discussion of the problem and a review of an Alaska experience. Alaska Med 15:27, 1973.

Mills WJ Jr, Gower R, Hackett P et al. Cold injury dehydration and multiple system trauma on Mt McKinley in hypoxia and cold. Sutton J, Houston C, Coates G (Ed.). 1987 340 Praeger NY.
Mills WJ Jr. Out in the cold. Emerg Med 8:134, 1975.
Mills WJ Jr. Field care of the hypothermic patient. Int J Sports Med Suppl 1:199, 1992.
Molnar GW. Survival of hypothermia by men immersed in the ocean. JAMA 131:1046, 1946.
Moore EE, Mattox KL, Feliciano DV. Trauma. 1991. Appletion and Lange. Conneticut.
Moore JB, Moore EE, Markovichick VJ et al. Diagnostic peritoneal lavage for trauma. Superiority of the open technique at the infra umbilical ring. J Trauma 21:570, 1981.
Morales CF, Strollo PJ. Non-cardiogenic pulmonary edema associated with accidental hypothermia. Chest 103:971, 1993.
Moritz AR, Henriques FC, McLean R. The effects of inhaled heat on the air passages and lungs: an experimental investigation. Am J Pathol 21:311, 1945.
Morris DL et al. Hemodynamic characteristics of patients with hypothermia due to occult infection and other causes. Ann Intern Med 102:153, 1985.
Morris DL, Jande MA. Diagnosis of sepsis in the hypothermic patient. J Clin Res 30:519A, 1982.
Morrison JB, Conn ML, Hayward JS. Thermal increment provided by inhalation rewarming from hypothermia. J Appl Physiol 46:1061, 1979.
Morrison JB, Conn ML, Hayes PA. Influence of respiratory heat transfer on thermogenesis and heat storage after cold immersion. Clin Sci 63:127, 1982.
Motin J et al. L'hypothermie accidentielle au cours des intoxications par less neuroleptiques et les tranquillis sants. Lyon Med 230:53, 1973.
Mouritzen CV, Anderson MN. Myocardial temperature gradients and ventricular fibrillation during hypothermia. J Thorac Cardiovasc Surg 49:937, 1965.
Murphy JV, Banwell PE, Roberts AH, McGrouther DA. Frostbite: pathogenesis and treatment. J Trauma Jan:48(1):171, 2000.
Myers RA, Britten JS, Cowley RA. Hypothermia: quantitative aspects of therapy. JACEP 8:523, 1979.
Nahun LH, Phillips R. The effect of hypothermia on digitalis intoxication. Conn Med 23:568, 1959.
Naucler S. Proceedings of the Royal Swedish Academy of Sciences. 1756.
Neil HA, Dawson JA, Baker JE. Risk of hypothermia in elderly patients with diabetes. Br Med J 293:416, 1986.

Niazi SA, Lewis FJ. Profound hypothermia in man: report of a case. Ann Surg 147:264, 1958.
Nielson KC, Owman C. Control of ventricular fibrillation during induced hypothermia in cats after blocking the adrenergic neurons with bretylium. Life Sci 7:159, 1968.
Nissen ER et al. A case of bullous frostbite following recreational snowmobiling. Cutis 63:21, 1999.
Nose H. Transvascular fluid shift and redistribution of blood in hypothermia. Jpn J Physiol 32:831, 1982.
Nozaki R et al. Accidental profound hypothermia. N Engl J Med 315:1680, 1986.
O'Brien H, Amess JA, Mollin DR. Recurrent thrombocytopenia, erythroid hypoplasia, and sideroblastic anemia associated with hypothermia. Br J Haematol 51:451, 1982.
O'Bryne P et al. Treatment of severe accidental hypothermia with a simple extracorporeal circuit. Agressologie 30:541, 1989.
O'Connor JP. Use of peritoneal dialysis in severely hypothermic patients. Ann Emerg Med 15:104, 1986.
O'Keefe KM. Accidental hypothermia: a review of 62 cases. JACEP 6:491, 1977.
O'Malley J, Mills W, Kappes B, Sullivans S. Frostbite: general and specific treatment. The Alaska Method. Algorithm for treatment. Providence Hopsital, Anchorage AL, 1993.
O'Sullivan ST et al. Baron Larrey and cold injury during the campaign of Napoleon. Ann Plast Surg 34:446, 1995.
Okada M. The cardiac rhythm in accidental hypothermia. J Electrocardiol 17:123, 1984.
Okada M, Nishimura F. Respiratory function and acid-base status in accidental hypothermia assessed by arterial blood gas analysis. Jpn J Med 29:500, 1990.
Okuboye JA, Ferguson CC. The use of hyperbaric oxygen in the treatment of experimental frostbite.Canad J Surg. 1968;11:78..
Orr KD, Fainer DC. Cold injuries in Korea during the winter of 1950-51. Medicine 1952:31:177, 1952.
Osczevski RJ, The Basis of Wind Cill. Arctic 48:372, 1995.
Oung CM et al. Effects of hypothermia on hemodynamic responses to dopamine and dobutamine. J Trauma 33:671, 1992.
Pace NL. A critique of flow-directed pulmonary arterial cathterization. Anesthesiology 47:455, 1977.
Paton BC. Accidental hypothermia. Pharmacol Ther 22:331, 1983.
Paton BC. A history of frostbite treatment. Int J Circumpolar Health 2000:59:99.

Perlstein PH et al. Adaption to cold in the first three days of life. Pediatrics 54:411, 1974.
Pickering BG, Bristow GD, Craig DB. Case history number 97: core rewarming by peritoneal irrigation in accidental hypothermia with cardiac arrest. Anesth Analg 56:574, 1977.
Pina-Cabral JM, Ribeiro-da-Silva A, Almeida-Dias A. Platelet sequestration during hypothermia in dogs treated with sulphinpyrazone and ticlopidine: reversibility accelerated after intra-abdominal rewarming. Thromb Haemost 54:838, 1985.
Pinzur MS, Weaver FM. Is urban frostbite a psychiatric disorder? Orthopedics Jan:20(1):43, 1997.
Pittet D et al. Bedside prediction of mortality from bacteremic sepsis. A dynamic analysis of ICU patients. Amer J Respir Crit Care Med 153:684, 1996.
Pledger HG. Disorders of temperature regulation in acute traumatic tetraplegia. J Bone Joint Surg 44:110, 1963.
Preston BR. Effect of hypothermia on systemic and organ system metabolism and function. J Surg Res 20:49, 1976.
Pugh L, Edholm O, et al. A physiological study of channel swimming. Clin Sci 19:257, 1960.
Rahn H, Reeves RB, Howell BJ. Hydrogen ion regulation, temperature, and evolution. Am Rev Respir Dis 112:165, 1975.
Rahn H. Body temperature and acid-base regulation. Pneumonologie 151:87, 1974.
Rankin AC, Rae AP. Cardiac arrhythmias during rewarming of patients with accidental hypothermia. Br Med J 289:784, 1984.
Ream AK, Reitz BA, Silverberg G. Temperature correction of $PaCO_2$ and pH in estimating acid-base status: an example of emperor's new clothes? Anesthesiology 56:41, 1982.
Reed RL et al. Hypothermia and blood coagulation: dissociation between enzyme activity and clotting factor levels. Circ Shock 32:141, 1990.
Reed RL et al. The disparity between hypothermic coagulopathy and clotting studies. J Trauma 33:465, 1992.
Reisinger KS, Kao J, Grant DM. Inaccuracy of the Clinitemp skin thermometer. Pediatrics 64:4, 1979.
Reuler JB, Jones SR, Girard DE. Hypothermia in the erythroderma syndrome. West J Med 127:243, 1977.
Reuler JB, Parker RA. Peritoneal dialysis in the management of hypothermia. JAMA 240:2289, 1978.
Reuler JB. Hypothermia: pathophysiology, clinical settings, and management, Ann Intern Med 89:519, 1978.
Robinson M, Seward PN. Environmental hypothermia in children. Pediatr Emerg Care 2:254, 1986.

Rodrigo N, Ranwala R. Delayed recovery following atracurium: case report. Eur J Anaesthosiol 5:207, 1988.
Rogers IR et al. Infrared emission tympanic thermometers cannot be relied upon in a wilderness setting. Wilderness and Environ Med 10:201, 1999.
Rohrer MJ, Natale AM. Effect of hypothermia on the coagulation cascade. Crit Care Med 20:1402, 1992.
Romet TT. Hoskin RW. Temperature and metabolic responses to inhalation and bath rewarming protocols. Aviat Space Environ Med 59:630, 1988.
Rosenkrantz L. Bone marrow failure and pancytopenia in two patients with hypothermia. South Med J 78:358, 1985.
Sabapathi R, Ridley C, Yen MC. Complete recovery from profound hypothermia associated with DIC. Md Med J 35:203, 1986.
Sampson JB. Anxiety as a factor in the incidence of combat cold injury: a review. Milit Med 149:89, 1984.
Schaller MD, Fisher AP, Perret CH. Hyperkalemia: a prognostic factor during acute severe hypothermia. JAMA 264:1842, 1990.
Schissel DJ et al. Cold weather injuries in an arctic environment. Mil Med 163:568, 1998.
Sekar TS et al. Survival after prolonged submersion in cold water without neurological sequelae. Arch Intem Med 140:775, 1980.
Severinghaus JW, Astrup PB. History of blood gas analysis. III. Carbon dioxide tension. J Clin Monit 2:60, 1986.
Severinghaus JW. Blood gas calculator. J Appl Physiol 21:1108, 1966.
Shenaq SA et al. Effect of profound hypothermia on leukocytes and platelets. Ann Clin Lab Sci 16:130, 1986.
Shields CP, Sixsmith DM. Treatment of moderate-to-severe hypothermia in an urban setting. Ann Emerg Med 19:1093, 1990.
Siebke H, Breivik H, Rod T et al. Survival after 40 minutes' submersion without cerebral sequelae. The Lancet 1:1275, 1975 .
Sinet M et al. Maintaining blood pH at 7.4 during hypothermia has no significant effect on work of the isolated rat heart. Anesthesiology 62:582, 1985.
Siple P, Passel CF. Measurements of dry atmospheric cooling in subfreezing temperatures. Proc Am Philosoph Soc 89:177,1945.
Smith M et al. Injuries in cross-country skiing: a critical appraisal of the literature. Sports Med 21:239, 1996.
Smith RM, Hanna JM. Skinfolds and resisting heat loss in cold air and water temperature equivalence. J Appl Physiol 39:93,1975.
Sofer S, Benkovich E. Severe infantile hypothermia: short- and long-term outcomes. Intensive Care Med 26:88, 2000.

Soung LS et al. Treatment of accidental hypothermia with peritoneal dialysis. Can Med Assoc J 117:1415, 1977.
Southwick FS, Dalglish RH. Recovery after prolonged hypothermia. JAMA 243:1250, 1980.
Spelman E. Xenophon: The Expedition of Cyrus into Persia, and the Retreat of Ten Thousand Greeks, 400 B.C. Translated, Esq (2 vols.). London. 1749 D. Brown et al.
Statistics Sweden. Register of causes of death, 1994-1998, 2000.
Steinman A. Prehospital management of hypothermia. Response 6:18, 1987.
Steinman A. Cold water immersion, in Wilderness Medicine, Auerbach P, 4:104, 1995, ed. Mosby, 1995, 104.
Steinmann S, Shackford SR, Davis JW. Implications of admission hypothermia in trauma patients. J Trauma 30:200, 1990.
Sung Y et al. Susceptibility of human and porcine neutrophils to hypothermia in vitro. Pediatr Res 19:1044, 1985.
Swain JA, White FN, Peters RM. The effect of pH on the hypothermic ventricular fibrillation threshold. J Thorac Cardiovasc Surg 87:445, 1984.
Swain JA. Hypothermia and blood pH: a review. Arch Intern Med 148:1643, 1988.
Swain JA et al. Hemodynamics and metabolism during surface induced hypothermia in the dog: a comparison of pH management strategies. J Surg Res 48:217, 1990.
Swan H et al. Cessation of circulation in general hypothermia. I. Physiologic changes and their control. Ann Surg 138:360, 1953.
Swartz RD et al. Hypothermia in the uremic patient. Dialysis Transplant 12:584, 1983.
Szekely P, Wynne NA. The effects of digitalis on the hypothermic heart. Br Heart J 22:647, 1960.
Tan S. Rewarming after cardiopulmonary bypass: a comparison of two methods. Ann Acad Med Singapore 19:350, 1990.
Taylor MS. Lumbar epidural sympathectomy for frostbite injuries of the feet. Mil Med 164:566, 1999.
Tikuisis P. Predicting survival times at sea based on observed body cooling rates. Aviat Space and Environ Med 68:441, 1997.
Tipton M et al. Immersion deaths and deterioration in swimming performance in cold water. Lancet 354:626, 1999.
Tisherman SA et al. Profound hypothermia (less than 10°C) compared with deep hypothermia (15°C) improves neurological outcome in dogs after two hours' circulatory arrest induced to enable resuscitative surgery. J Trauma 31:1051, 1991.

Wallace W. Does it makes sense to heat gases higher than body temperature for the treatment of cold water near-drowning or hypothermia? A point of view paper. Alaska Med 39: 75, 87, 1997.
Valnicek SM et al. Frostbite in the prairies: a 12 year review. Plast and Reconstruct Surg 92:633, 1993.
Walpoth BH et al. Assessment of hypothermia with a new tympanic thermometer. J Clin Monit 10:91, 1994.
Walpoth BH, et al. Outcome of survivors of accidental hypothermia and circulatory arrest treated with extracorporeal blood warming. New Eng J Med 337:1500, 1997.
Vandam LD, Brunap TK. Hypothermia. N Engl J Med 261:546, 1959.
Wang LS et al. A re-evaluation of heparin requirements for cardiopulmonary bypass. J Thorac Cardiovasc Surg 101:153, 1991.
Vangaard L, Gjerloff CC. A new simple technique of rewarming in hypothermia. Int Rev Army Navy Air Force Med Serv. 52:427, 1979.
Vangaard L. The physiology of hypothermia. Blood flow in hands and feet. Arterio anastomoses and the ”heating glow”. Reported by Mills W and Pozos R. Low temperature effects on humans. Encyclopedia of Human Biology. 1990 Academic Press.
Webb GE. Comparison of esophageal and tympanic temperature monitoring during cardiopulmonary bypass. Anesth Analg 52:729, 1973.
Wedin B, Vanggard L, Hirvonen J. ”Paradoxical undressing” in fatal hypothermia. J Forensic Sci 24:543, 1979.
Weisfeldt ML, Chandra N, Tsilik J. Increased intrathoracic pressure-not direct heart compression-causes the rise in intrathoracic vascular pressures during CPR in dogs and pigs. Crit Care Med 9:377, 1981.
Wessel HU, James GW, Paul MH. Effects of respiration and circulation on central blood temperature of the dog. Am J Physiol 211:1403, 1966
Westin B, Sehgal N, Assali NS. Regional blood flow and vascular resistance during hypothermia in dogs. Am J Physiol 201:485, 1961.
White JD. Hypothermia: the Bellevue experience. Ann Emerg Med 11:417, 1982.
White JD et al. Comparison of rewarming by radio wave regional hypothermia and warm humidified inhalation. Aviat Space Environ Med 55:1103, 1984.
White JD et al. Controlled comparison of radio wave regional hyperthermia and peritoneal lavage-rewarming after immersion hypothermia. J Trauma 25:989, 1985.
White JD et al. Rewarming in accidental hypothermia: radio wave versus inhalation therapy. Ann Emerg Med 16:50, 1987.
Wilkerson J, Hamlet MP. Medical after action conference, Mount Hood, 1986, bypass rewarming. US Army research institute of environmental medicine, 1988.

von Segesser LK, Garcia E, Turina M. Perfusion without systemic heparinization for rewarming in accidental hypothermia. Ann Thorac Surg 52:560, 1991.
von Segesser LK et al. Reduction and elimination of systemic heparinization during cardiopulmonary bypass. J Thorac Cardiovasc Surg 103:790, 1992.
Voorhees WD, Babbs CF, Tacker WA. Regional blood flow during cardiopulmonary resuscitation in dogs. Crit Care Med 8:134, 1980.
Wrenn K. Immersion foot. A problem of the homeless in the 1990's. Arch Int Med 151:785, 1991.
Vretenar DF et al. Cardiopulmonary bypass resuscitation for accidental hypothermia. Ann Thorac Surg 58:895, 1994.
Young AJ. Effects of aging on human cold tolerance. Exp Aging Res 17:205,1991.
Zook N et al. Microcirculatory studies of frostbite injury. Ann Plastic Surg 40:246, 1998.

Near-drowning

Dr *Murray Hamlet*, formerly of the US Army Research Institute of Environmental Medicine, Natick MA, USA.

Introduction

Near drowning is a significant physiologic insult. It is an emotionally charged situation at the rescue, during transport, and in the emergency room. Everyone is affected by the incident and should be recognised as a partial victim of the episode after the treatment process is completed.

There is relatively little scientific information available on the drowning process (Golden FS et al 1997). Most of the work has been done on dogs and it is only recently that we understand that dogs probably do not go through the same drowning scenario as humans (Modell JH et al 1976). Physicians tend to extrapolate other treatment approaches to drowning, such as treatment protocols for traumatic brain injuries, which may or may not be appropriate. Adding to this problem, the outcomes vary widely even in relatively similar submersion experiences. This variability in neurological survival after submersion complicates the recommendations for treatment and the prognostic capability during treatment. There have been some successes after long-time submersion with excellent neurological results, but there have been many more failures even with shorter submersion times. In addition, there is significant controversy about treatment, which will be covered later in this discussion.

Immersion and submersion

It is important to define the difference between immersion and submersion. These terms are commonly used interchangeably, which leads to significant confusion. “Immersion” means that an individual has been immersed in water, but the airway was still above the water’s surface. “Submersion” is the best term to describe near drowning, and occurs when the individual, including the airway, has been totally under water.

Cold water

It is important to define cold water. It has been shown that cold protects the individual during drowning (Norberg WJ et al 1992, Schmidt U et al 1995). Cold decreases the metabolic demands of the tissues, and in particular the brain (Modell JH 1971, 1986). While cold decreases metabolic demands it also increases the likelihood of survival (Bierens JJ et al 1990). A consensus meeting of experts in hypothermia and drowning in Winnipeg, Canada defined water colder than 10 °C as "cold water." You can expect some chance of survival if the water is colder than 10 °C (Conn AW 1976; Modell JH et al 1976; Orlowski JP 1979; Huckabee HC et al 1996).

Epidemiology

Drowning is a severe universal problem. Every year approximately 300 000 persons drown worldwide. This implies that someone drowns every second minute. In Sweden, with 9 million inhabitants, someone dies from drowning every third day. At the end of the 19th century about 1 000 people drowned in Sweden every year (19,6 per 100 000 inhabitants). During 1940–1950 a great effort was made to teach people how to swim and the number of deaths by drowning went down to 500 per year (8,2 per 100 000 inhabitants). This diminishing trend has continued as more and more people have learnt to swim and the annual mortality figure for 1991–2000 is about 150 per year. (1,7 per 100 000 inhabitants).

Males are much more commonly involved in drowning accidents than females. In Sweden during 2000 91 % of those who drowned were men. In 1999 79 % were men. Risky outdoor activities and an inclination amongst men to take more risks might explain this imbalance.

People more than fifty years old are over-represented among drowning victims. This may be because this age group did not have the same opportunities to learn how to swim while growing up, but also to an inability to withstand the physical stress involved in drowning. Drowning is the second leading cause of accidental death amongst children in Sweden, only surpassed by trauma. According to the Swedish Lifesaving Society seven out of ten adults who drown are under the influence of alcohol.

The drowning statistics in the United States are somewhat similar to those in Sweden. The annual death rate from drowning is 1.65/100.000, but the rate in Alaska is much higher than the national average: 7.41/100,000. The very high rate in Alaska is due to the high mortality among sea fishermen. Falling off a fishing boat in the seas around Alaska is almost a sentence of death due to the low water temperature and the rough seas. As in Sweden most of the casualties (81 %) occur in men. Fifty

percent (50 %) of drowning accidents in adolescent boys are associated with alcohol consumption and 25–50 % of deaths associated with boating accidents are alcohol related. Drowning is the second most common cause of traumatic death in small children and most of the deaths occur in swimming pools (National Center for Health Statistics 2000). People drown in many different settings, especially in outdoor activities during the spring, summer and early autumn. The most common scenarios involve outdoor swimming, falling from small boats often close to the shore and in accidents while hiking, skating or snowmobiling on thin ice.

Pathophysiology

The following description applies to drowning in both cold and warm water. There is little difference in the pathophysiology except that the chances of survival are improved in victims who are submerged in cold water because of the neurological protection from hypothermia. The metabolic and physiologic changes in hypothermia that have been described in earlier sections of the main report apply equally to the victim who is hypothermic because of submersion.

When a person is plunged into cold water, be it out of a boat or through the ice, there is an initial gasp reflex. If the victims sink so quickly that they are underwater when this gasp reflex occurs, they aspirate cold water (Longheed DW et al 1939). At this point, ventilation increases fivefold. This hyperventilation decreases the $PaCO_2$, which may lead to tetany. Breath holding capability decreases with cold water submersion. Peripheral vasoconstriction leads to a decrease in muscle blood flow. There is an increase in systolic blood pressure, (from normally 130 up to 180–190) secondary to an outpouring of adrenaline. Cardiac output increases from 5 up to 10 litres per minute, which is double the normal, atrial and ventricular extra systoles can lead to cardiac arrest (Keatinge WR and Hayward MG 1981), or ventricular fibrillation. All these arrhytmias can result from myocardial irritability secondary to rapid pH changes, hypoxia, hypo- and hyperkalaemia. Prolonged immersion, such as floating in a life jacket, without fluid intake and with an associated cold diuresis, can produce hypovolemia.

Not all submersion victims aspirate water. Ten to fifteen percent have dry lungs (Modell JH 1971). Whether from laryngeal spasm or from rapid absorption of small volumes of water, the lungs appear to be dry. At first glance, it may appear that these victims would have a better chance of survival, but this has not been proven to be so. The total hypoxic time, whether due to aspiration of water or laryngeal spasm, is the critical factor in determining survival.

Animal models show that the cardiac lesions after submersion are similar to the those in hearts exposed to high levels of catecholamines (Eliot RS et al 1979; Karch SB 1986). The high stress of drowning may produce these lesions. Animals often continue to breathe under water after submersion. Interviews with survivors of drowning have revealed that some humans do so too. Continued breathing underwater may have protective effects that will be described later.

Submersion in fresh water

Aspiration of fresh water is thought to lead to some haemolysis and hyperkalaemia (Modell JH and Moya F 1966). As water leaves the alveolus and enters the general circulation, there appears to be a relative increase in blood volume. Fresh water, hypotonic in relation to plasma, dilutes the blood and increases the intravascular volume (Conn AW 1979, Conn AW and Barker GA 1984). Osmosis drives free water into the red cells that swell and lyse, leading to the release of haemoglobin and intracellular potassium into the serum. Serum potassium levels rise. Simultaneously, there is a relative decrease in serum calcium and magnesium. Alveolar surfactant is washed out, leading to a ventilation-perfusion imbalance and hypoxia (Modell JH et al 1968). It is interesting to note however, that very few survivors have specific major electrolyte changes.

Submersion in salt water

Salt-water submersion causes plasma fluid to move into the alveoli and pulmonary tissue (Giammonna ST and Modell JH 1967). The osmolarity of seawater is three or four times that of blood which means that it pulls a protein rich fluid from the blood and into the alveolus (Modell JH et al 1967). While air and water pass in and out of the airways, a thick foamy proteinaceous material is created, threatening the ventilation efforts of the victim (Greenberg MI et al 1982): Following this there is also an apparent decrease in blood volume and an increase in plasma electrolytes. This is followed by a decrease in the cough reflex and an increase in parenchymal pulmonary damage.

The end result in both fresh and salt water is progressive hypoxemia.

Survival factors

Although the numbers are few, there are several records of victims who survived, neurologicalally intact after one-hour of submersion (Fields AI 1992). There are many survivors of a half-hour submersion, but from a half-hour to an hour, survival potential decreases dramatically. Two factors play a role in these survivors (Elixson EM 1991; Gooden BA 1992),

the first is the dive reflex (Hayward JS et al 1984; Gooden BA 1992), a vagally mediated reflex that occurs when the face and forehead are cooled and the face is submerged in water. There is significant bradycardia, and shunting of blood from the periphery and central organs to cerebral flow. The brain-heart-lung circulatory loop is maintained at the expense of the rest of the body. This reflex is protective and allows for feeding behaviours in a variety of mammals, such as seals, but may also play a role in difficult parturition. The second is that when the dive reflex is in place, there is significant internal cooling with continued respiration under water that floods the lungs with cold water that is subsequently absorbed into the central circulation. Animal modelling of this cooling mode explains the significant loss of core temperature in some submerged children (Giammonna ST 1971).

There has been some question whether people in cold water die of hypothermia or drowning. Almost all die of drowning. The key is whether the flotation device they are wearing will hold their head above water when they are unconscious. If it will not, then most drown. If it will, they will lose consciousness and then, some time later, depending on body size, thickness of subcutaneous fat, insulation of clothes and temperature and condition of waters, they will die of hypothermia. Hypothermia, however, complicates survival in that the victims quickly loose the ability to co-ordinate their swimming motions or to have any grip strength. They are unable to do many of the things that are necessary for survival in cold water, including hanging on to other victims, wreckage, or an overturned boat.

Treatment

Pre-Hospital Treatment

It is generally believed that if submersion is documented for 60 minutes or longer, CPR and resuscitation should not be attempted. Even though there have been survivors after 66 minutes of submersion (Bolte RG 1988), the majority of individuals submerged for an hour or longer will have a major neurological deficit, no matter how they are treated. Also, if there are associated injuries, such that they cannot survive, or they are asystolic as a result of trauma, then resuscitation should not be attempted.

Pre-hospital treatment includes getting to the patient, clearing their airway, and preventing aspiration from vomiting (Giammonna ST 1971; Emergency Cardiac Care Committee and Subcommittees 1992; Kyriacou DN et al 1994). Ventilate the patient and oxygenate with 100 % O_2 with positive end-expiratory pressure (PEEP). Intubate if necessary. If there is no pulse, start CPR. A patient submerged in fresh water may

show a different picture from one submerged in saltwater. The former has little or no water in the airway: the latter has thick foamy sputum. Therefore, be sure to have a good suction device available in the latter case and be prepared for more difficulty in clearing the airway. Initiate CPR, if indicated. All submersion victims, even if conscious and lucid, should be hospitalised and observed for at least 4 to 6 hours. Emergency medical personnel should be ready to provide the physicians with on-scene information such as the duration of submersion, the temperature of the water, the time to initiate resuscitation after rescue, the duration of transport, any trauma information and the responsiveness of the patient during the transport. Precise information on how the patient was found – partially conscious with heart and lung functioning, or flaccid, comatose with fixed and dilated pupils, or asystolic, will add to the physician's ability to decide whether to continue treatment or terminate the process.

Hospital Management

Asymptomatic patients

Observe asymptomatic patients for at least 4 to 6 hours in the Emergency Department. Take chest x-rays, administer supplemental oxygen, and draw blood for gasses, ethanol and a toxicology screen. Secondary pulmonary changes can occur hours after submersion. Make sure these complications occur in the hospital, and not at home because the patient was discharged too soon.

Symptomatic patients

Any patient who shows signs of distress (anxiety, tachypnea, dyspnea, syncope, persistent cough, or vital sign change) should receive high flow oxygen, 10–15 l/min on a non-rebreathing mask and be intubated if necessary. Measure the blood gasses and pH, take chest x-rays, and insert a stomach tube to drain both air and stomach contents. Careful management of the airway is extremely important, as vomiting and aspiration are common. Bronchial dilators may be helpful in management of bronchial constriction or bronchial irritation associated with submersion.

The comatose, arrested patient is the real challenge. Intubate immediately and ventilate with 100 % oxygen with PEEP, start CPR (Norberg WJ et al 1992; Schmidt U et al 1995). If the patient is clearly hypothermic reduce the frequency of ventilation and CPR to compensate for actual body temperature. In the initial phase try to attain a PaO_2 of 150 mm Hg (20 kPa), to minimise the risk of hypoxia ,and PaC_2O of 30 mm Hg (4 kPa). In a later phase the most desirable PaO2 and PaCO2 must be individually chosen according to the severity of the pulmonary and cerebral insult in the specific patient. If the arterial pH is less than 7.25, administer a small amount of sodium bicarbonate. Culture the sputum. Consider

placing a Swan-Ganz catheter. It is generally wiser to use fluids to maintain cardiac output rather than pressor agents. If pressors are needed, Dopamine and Dobutamine will help. Give mannitol to maintain urine flow and decrease intracranial pressure. Calcium channel blockers may be helpful in preventing neurological damage, although this treatment is experimental. Cortico-steroids have not been proven to be as effective as first thought in the management of drowning victims.

The urgency of resuscitation is the same in hypothermic and normothermic patients, but if a cold patient is rewarmed rapidly before the blood and fluid volumes have been restored, hypotension will result. Respiration, fluid, electrolyte and acid-base balance must all be treated while initiating rewarming. Most submersion patients will not have serious depletion of blood volume because the exposure time is usually short. But if the patient has been exposed for a long time – as after a shipping accident – there may be serious volume depletion to take into account.

Prognostic factors

The worst environment in which to drown, by far, is raw sewage. Drowning accidents during the pumping of septic systems, and commercial diving in sewers, are almost universally fatal. Aspirating petroleum products on water after a shipwreck is also almost always fatal. Aspirating other chemical agents, solvents and foreign matter, particularly sand, also has a bad outcome (Hunter TB and Whitehouse WM 1974). Chlorine from chlorinated swimming pools was thought to be a problem, but it is now known that it is the hypotonicity of the water that is the biggest danger (Karch SB 1986). In addition, chlorine stuns, but does not kill, some organisms that could begin to grow in the lungs at a later stage when given the opportunity. When first cultured they may be thought to be saprophytes. These cultures should be kept, and if pneumonia develops, antibiotic sensitivity should be tested on them (Ender PT and Dolan MJ 1997). Thick algae from stagnant, derelict pools are also highly destructive to lung function.

The process for determining the prognosis is important (Modell JH 1971; Conn AW et al 1979). Part of the problem is that there is a wide range of response to treatment, and early prognosticators may not be accurate in determining eventual outcome (Modell JH 1986). Because of this wide variability, it is often appropriate to "give them a chance" despite a number of significant negative outcome criteria. Orlowski's criteria for unfavourable prognostic factors include: three years of age or less (Fields AI 1992) submersion time longer than five minutes, no resuscitation attempted for at least ten minutes after rescue, patient is in a coma upon admission to Emergency Department and arterial blood gas is pH is 7.1 or less. Others include: a Glasgow Coma Scale score of less than 5

and a submersion time of more than ten minutes (Dean MJ and Kaufman ND 1981). Any three criteria indicate a grave prognosis. Orlowski states that if three or more adverse criteria are present, there will be less than 5 % recovery (Orlowski JP 1987; Orlowski JP et al 1989; Quan L 1993). Good prognostic signs for field personnel include a rapid return of respiration, restoration of cardiac activity, improved oxygenation, positively responsive pupils and returning consciousness (Conn AW et al 1980). Good prognostic conditions are: the colder the water the better (Suominen et al 1997), the shorter the submersion, the quicker the onset of resuscitation, and the shorter the evacuation time. These criteria should be used both by pre-hospital and hospital management personnel to determine the continuation of treatment (Modell JH et al 1976).

The Controversies

Warm water drowning usually has a grim outcome. In cold water drowning hypothermia plays a role in survivability after submersion (Conn AW 1976; Biggart MJ et al 1990; Pearn J 1992; Golden 1997) and so far there is no controversy over this assertation. Controversy exists, on the other hand as to how survival is affected; is it due to cooling of the brain? Or to the dive reflex? The fact is there is wide variability in survival outcomes from similar submersion episodes in cold water (Bohn DJ et al 1986). Conductive cooling of the small head may play a role in children, but mathematically does not account for cooling as seen. Inhalation of cold water, however, has been mathematically modelled to increase both depth and rate of cooling. Therefore continued ventilation with cold water, with the dive reflex in place, may maintain selective cerebral circulation that could produce enough hypothermia to be protective. Even a decrease in brain temperature of 2 °C or 3 °C could have a significant protective effect, as oxygen requirement is reduced by 6–10 % per degree Celsius reduction in temperature. Although the dive reflex, with its selective adjustment of cerebral circulation and peripheral organ perfusion, may be quite variable in individuals, it has a protective effect which may be helpful in cerebral survival. This variability, along with the differences in onset and release of laryngeal spasm and body mass, may account for the dramatic variability in survival of submersion victims.

Most physicians now consider the cerebral damage of submersion to be a reperfusion injury. Sudden ischemia and delayed return of flow produce massive cytokine release and vascular collapse with increased endothelial permeability leading to progressive, rapid cerebral oedema. The same process occurs in the lungs, with increased permeability and pulmonary oedema. Sudden cooling of the lungs and brain appear to blunt the vascular effects and seems to be protective after submersion.

There is significant controversy over the use of extracorporeal perfusion (ECP) in submersion victims (Lavelle JM and Shaw KN 1980; Biggart MJ et al 1990; Lewis LM et al 1990; Walpoth BH et al 1990; Letsou GV et al 1992; Norberg WJ et al 1992; Kallas HJ and O´Rourke PP 1993; Quan L 1993; Waters DJ et al 1994). One side says there is a high likelihood of brain damage following resuscitation in the Emergency Room of an asystolic, drowning victim. Heart and lung function returns only to leave a live, brain-damaged child (Allman FD et al 1986; American Heart Association 1986). The other side say that asystolic submersion victims of cold water need every chance of survival that bypass offers.

ECP has everything the child needs: oxygenation, low-pressure circulation to the head, and maintenance of a cold temperature, while retaining cerebral perfusion. It both dilutes and removes potassium, and various compartment fluid management schemes can be conducted, all of which are critical elements in survival.

The speed of application, whether to perfuse a patient and selection of cessation criteria, are all fertile areas for research in this field. As with other medical emergencies, the decision may relate to the selection criteria for by-pass and to the techniques involved. Submersion time, age of the victim, temperature of the water, cardiac function at the time of rescue, and rescue time all play a role in neurological survival. Utilising these criteria, an algorithm for opting for by-pass, has not yet been constructed but ought to be.

When the patient enters the hospital the pump team has to be ready to make a decision to treat or not treat. The decision has to be made quickly, as the patient has no time to wait. There have been a number of highly publicised patients successfully managed with ECP that lead the public to believe that almost any cold-water submersion victim can be saved. This medical, legal, and ethical issue will not be resolved easily or quickly. Clear identification of selection criteria prior to cardiac by-pass is critical for a rational decision about which patients to receive this treatment (Kemp AM and Sibert JR 1991).

Criteria for Ceasing Resuscitation

If a patient meets three or more of Orlowski's criteria, receives **CPR** and is warmer than 35 °C, but has no cardiac or neurological response you can cease resuscitation efforts after 30 minutes because neurological damage is certain. This criterion corresponds with the standards of the US Coast Guard. If the patient is hypothermic and has been submerged less than 60 minutes, carry out CPR while rewarming the patient. If the patient is rewarmed to 35 °C in the ICU and is still unconscious after 24 hours or more, test to see if the patient has cerebral circulation or not.

The criteria for stopping treatment are failure to respond neurologically with no pupillary responses, non-awakening from a coma and the lack of cerebral reflexes. If however, the patient responds, continue treatment. If there are signs of a poor outcome and serious and obvious neurological damage, re-evaluate the patient every 12 hours to decide whether or not to maintain full intensive care.

References

Allman FD et al. Outcome flowing cardiopulmonary resuscitation in severe paediatric near drowning, *Am J Dis Child* 140:571, 1986.

American Heart Association. Standards and guidelines for cardiopulmonary resuscitation and emergency cardiac care, *JAMA* 255:2841, 1986.

Bierens JJ, van der Velde EA, van Berkel M. Submersion in The Netherlands: prognostic indicators and results of resuscitation, *Ann Emerg Med*, Dec 19(12):1390, 1990.

Biggart MJ, Bahn DJ, Desmond J. Effect of hypothermia and cardiac arrest on outcome of near-drowning accidents in children, *J Pediatr* 117:179, 1990.

Bohn DJ et al. Influence of hypothermia, barbiturate therapy, and intracranial pressure monitoring on morbidity and mortality after near drowning. *Crit Care Med* 14:529, 1986.

Bolte RG et al. The use of extracorporeal rewarming in a child submerged for 66 minutes. *JAMA 260:377.1988*

Conn AW. The role of hypothermia in near drowning. In *Proceedings of the Cold Water Symposium*, Toronto, 1976, Royal Life-Saving Society of Canada.

Conn AW. Near-drowning and hypothermia (editorial), *Can Med Assoc J* 120:397, 1979.

Conn AW, Edmonds JF, Barker GA. Cerebral resuscitation in near drowning, *Pediatr Clin North Am* 26:691, 1979.

Conn AW et al. Cerebral salvage in near drowning following neurological classification by triage, *Can Anaesth Soc J* 27:201, 1980.

Conn AW, Barker GA. Freshwater drowning and near drowning – an update, *Can Anaesth Soc J* 31:S38, 1984.

Dean MJ, Kaufman ND. Prognostic indicators in pediatric near drowning: the Glasgow Coma Scale, *Crit Care Med* 9:536, 1981.

Eliot RS, Todd GL, Pieper GM. Pathophysiology of catecholamine-mediated myocardial damage, *J SC Med Assoc* 75:513, 1979.

Elixson EM. Hypothermia. Cold-water drowning, *Crit Care Nurs Clin North Am*, Jun 3(2):287–92, 1991.

Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. IV. Special resuscitation situations, *JAMA* 268(16):2242, 1992.

Ender PT, Dolan MJ. Pneumonia associated with near drowning, *Clin Infect Dis*, Oct 25(4): 896, 1997.

Fields AI. Near-drowning in the pediatric population, *Crit Care Clin 8(1): 113, 1992.*

Giammonna ST, Modell JH. Drowning by total immersion: effects on pulmonary surfactant of distilled water, isotonic saline, and seawater, *Am J Dis Child* 114:612, 1967.

Giammonna ST. Drowning: pathophysiology and management, *Curr Probl Pediatrics* 3:3, 1971.

Golden F. Mechanisms of body cooling in submersed victims [editorial], *Resuscitation*, Oct 35(2): 107, 1997.

Golden FS, Tipton MJ, Scott RC. Immersion, near-drowning and drowning, *Br J Anaesth*, Aug, 79(2): 214, 1997.

Gooden BA. Drowning and the diving reflex in man, *Med J Aust* 2:583, 1972.

Gooden BA. Why some people do not drown: hypothermia versus the diving response, *Med J Aust* 157(9): 629, 1992.

Greenberg MI et al. Effects of endotracheally administered distilled water and normal saline on the arterial blood gases of dogs, *Ann Emerg Med* 11:600, 1982.

Hayward JS et al. Temperature effect on the human dive response in relation to cold water near drowning, *J Appl Physiol* 56:202, 1984.

Huckabee HC, Craig PL, Williams JM. Near drowning in frigid water: a case study of a 31-year-old woman, *J Int Neuropsychol*, May 2(3): 256, 1996.

Hunter TB, Whitehouse WM. Freshwater near-drowning: radiological aspects, *Radiology* 112:51, 1974.

Kallas HJ, O'Rourke PP. Drowning and immersion injuries in children, *Curr Opin Pediatr*, Jun 5(3): 295, 1993.

Karch SB. Pathology of the heart in near-drowning, *Arch Pathol Lab Med* 109:176, 1985.

Karch SB. Pathology of the lung in near-drowning, *Am J Emerg Med* 4:4, 1986.

Keatinge WR, Hayward MG. Sudden death in cold water and ventricular arrhythmia, *J Forensic Sci* 26:459, 1981.

Kemp AM, Sibert JR. Outcome in children who nearly drown: a British Isles study, *BMJ*, Apr 20(6782): 931, 1991.

Kyriacou DN, Arcinue EL, Peek C, Kraus JF. Effect of immediate resuscitation on children with submersion injury, *Pediatrics*, Aug 94(2, Pt1): 137, 1994.
Lavelle JM, Shaw KN. Near drowning: is emergency department cardiopulmonary resuscitation or intensive care unit cerebral resuscitation indicated? *Crit Care Med* 21(3): 368, 1980.
Letsou GV et al. Is cardiopulmonary bypass effective for treatment of hypothermic arrest due to drowning or exposure? *Arch Surg*, May 127(5):525, 1992.
Lewis LM, Ruoff B, Rush C, Stothert JC Jr. Is emergency department resuscitation of out-of-hospital cardiac arrest victims who arrive pulseless worthwhile? [published erratum appears in *Am J Emerg Med*, Jul 8(4):371, 1990] *Am J Emerg Med*, Mar 8(2):118, 1990.
Longheed DW, James JM, Hall GE. Physiological studies in experimental asphyxia and drowning, *Can Med Assoc J* 40:423, 1939.
Modell JH, Moya F. Effects of volume of aspirated fluid during chlorinated fresh water drowning, *Anesthesiology* 27:633, 1966.
Modell JH et al. Effects of fluid volume in sea-water drowning. *Ann Int Med* 67:68, 1967.
Modell JH et al. Blood gas and electrolyte changes in human near-drowning victims. *JAMA* 203:99, 1968.
Modell JH, Graves SA, Ketover A. Clinical course of 91 consecutive near-drowning victims, *Chest* 10:231, 1976.
Modell JH. Pathophysiology and treatment of drowning and near-drowning, *Springfield, IL, , Charles C Thomas 1971.*
Modell JH. Treatment of near-drowning: is there a role for H.Y.P.E.R. therapy? *Crit Care Med* 14:593, 1986.
National Center for Health Statistics (NCHS), National Mortality Data, 1998. Hyattsville (MD) : NCHS 2000.
Norberg WJ et al. Successful resuscitation of a cold water submersion victim with the use of cardiopulmonary bypass, *Crit Care Med*, Sep 20(9): 1355, 1992.
Orlowski JP. Prognostic factors in drowning and near-drowning, *J Am Coll Emerg Phys* 8:176, 1979.
Orlowski JP. Drowning, near-drowning and ice-water submersion, *Pediatr Clin North Am* 34:75, 1987.
Orlowski JP et al. The hemodynamic and cardiovascular effects of near drowning in hypotonic, isotonic, or hypertonic solutions, *Ann Emerg Med* 18:1044, 1989.
Pearn J. Medical aspects of drowning in children, *Ann Acad Med Singapore*, May 21(3): 433, 1992.
Quan L. Drowning issues in resuscitation, *Ann Emerg Med* 22(2, Pt 2): 366, 1993.

Schmidt U, Fritz KW, Kasperczyk W, Tescherne H. Successful resuscitation of a child with severe hypothermia after cardiac arrest of 88 minutes, *Prehospital Disaster Med*, Jan-Mar 10(1): 60, 1995.

Suominen PK, Korpela RE, Silfvast TG, Olkkola K. Does water temperature affect outcome of nearly drowned children, *Resuscitation*, Oct 35(2): 111, 1997.

Walpoth BH et al. Accidental deep hypothermia with cardiopulmonary arrest: extracorporeal blood rewarming in 11 patients, *Eur J Cardiothorac Surg*, 9(7): 390, 1990.

Waters DJ, Belz M, Lawse D, Ulstad D. Portable cardiopulmonary bypass: resuscitation from prolonged ice-water submersion and asystole, *Ann Thorac Surg*, Apr 57(4): 1018, 1994.

General Reference

Newman AB. Submersion Incidents in Wilderness Medicine. 4th Edition. Auerbach PS (ed). Mosby, St.Louis 2001.

APPENDIX 2

Prevention of cold injuries

Ingvar Holmér, Professor of Climate Physiology, National Institute of Working Life, Stockholm, Sweden

What does "cold" mean?

Perceptions of cold, warmth and comfort temperature are highly subjective. They are coloured by experience, expectations and needs. An experienced mountain hiker will react and adjust to cold in a different way from the tyro.

Although the risk of problems increases as temperatures fall, it is not temperature alone that decides how difficult things are going to be. It is more enlightening to analyse the possibilities of creating good thermal balance and the exertions, compromises and sacrifices which this will require. Objectively speaking, cold stress is determined by an interaction of several climatic factors that create a motive force for the escape of heat from the body. The resultant heat lost is determined by the actions taken, consciously or unconsciously, by the individual, such as the choice and adaptation of clothing, protection and exposure times.

What are the problems?

Cold and a cold climate affect us in many ways. The following factors, singly or in various combinations, create a complex environment with an abnormally high risk of injuries and accidents.

- Cold creates a constant risk of losing thermal balance. The body or the hands and feet begin to feel cold. Wind chill or contact with cold objects can give rise to cold injuries.
- Cold imposes great mental strain. Concern for protecting oneself against the cold is added to involvement in the actual task in hand. Concentration, attention and decision-making have to be divided between a larger number of tasks.
- Protection from the cold solves certain problems but creates new ones. Thick, heavy clothing impedes movement and mobility. Gloves

and mittens reduce dexterity. Work becomes heavier and more complicated and progresses more slowly.
- The increased mechanisation of many jobs makes them easier but at the same time more sensitive to cold. Because less physical activity is required more protection is needed against cold.
- Snow, wind and ice cause problems with cold. In addition to heavier demands on personal protection, the cold causes more adverse conditions at the workplace.
- Cold, ice and snow affect the efficiency and safety of vehicles, machinery and tools.
- Present-day leisure habits, (in the mountains and at sea) involve rapid travel between urban communities and wilderness country, with an attendant risk of communication-related and logistical problems.
- Frequent alternations between a cold and normal climate require perpetual adjustments to clothing and working methods.

How are we affected by cold?

Cold affects the individual in two ways, directly through climatic impact, and indirectly through the effects of protective equipment.

- Increased heat losses lead to general and/or local hypothermia.
- Measures taken to keep out the cold entail compromises and restrictions affecting working methods and capacity.

One necessary precondition for safe exposure to and work in cold conditions is for the individual to be able to regulate and control his thermal balance, so that equilibrium is maintained and unnecessary heat losses are eliminated or restricted. Prevention is the best defence against hypothermia i.e. deliberate selection and modification of clothing and other equipment according to the circumstances. Deliberate actions also include the regulation of working pace and working hours. The combination of flexible clothing and variable activity is a strong enough measure to provide adjustment opportunities in a very wide range of temperature (Fig 1). Experience, knowledge and training coupled with good equipment, are necessary adjuncts to this process.

If the measures taken are insufficient to prevent hypothermia of the whole body or parts of it, a series of physiological reactions begins, aimed at limiting the effects of the cold and of protecting vital organs. This physiological heat control is automatic and works on the circulation, sweating or heat production (shivering) according to need. The result of this adjustment process determines the risk of cold injury occurring.

Climate, clothing, behaviour

Thermal balance

The risk of thermal imbalance and hypothermia is determined primarily by the interaction of climate, clothing and behaviour. Because climatic conditions are difficult to predict and influence, other factors have to be adapted to them. Appropriate choice and use of clothing and sensible behaviour in the cold are important components of this adjustment.

Thus the impact of climate on the body is determined by the possibilities of maintaining a balance between the body's own heat production and its heat emissions. The thermal balance can be described in the following equation:

$$M - W = RES + E + R + C + K + S$$

M is the body's energy production (metabolism), W is energy used for external mechanical work, RES is heat exchange through breathing (respiration), E is heat exchange through evaporation, R is heat exchange through radiation, C is heat exchange through convection, K is heat exchange through conduction and S is a possible change in the body's thermal content. M–W indicate the body's heat production. W, as a rule, is very small and can be disregarded. During uphill walking (in mountains, for example) W can be 10 or 15 per cent of M.

The body's own heat production (M–W) is determined by basic energy turnover and by muscular activity (in the form of work or shivering). Examples of the heat output of the body in connection with different activities will be found in Table 1.

Table 1. Examples of energy production during various sustained activities and shivering. 1 kcal/hour =1.16W

Activity	W	Kcal/hour
Sleeping	80–90	70–80
Sitting, standing	100–150	85–130
Light manual work	150–200	130–170
Easy walking, 3 km/hour	200–250	170–215
Heavy arm work	300–350	255–300
Fast walking, 5 km/hour	300–400	255–340
Heavy exertion with the whole body	400–600	340–500
Very heavy physical labour	600–900	500–800
Top-level sport: skiing, skating, running	1,200–2,000	1,000–1,700
Shivering during hypothermia	200–400	170–340

Heat exchange with surroundings is a physical process determined by known rules. Thermal energy is transferred from a warm surface (skin) to a colder medium. Heat is emitted through convection, radiation, con-

duction and evaporation from the surface of the body and through convection and evaporation from breathing.

Convection: Air is warmed by contact with the skin (or different layers of clothing). Warm air becomes lighter and therefore flows upwards and away from the body, ("chimney effect"). The stronger the air stream caused by wind or movement, the greater the amount of heat emitted. Air temperature and wind velocity are important determining factors.

Radiation: Heat is emitted through "wave movements" directly from the skin, (or surface of the clothing) to surrounding colder surfaces. (Compare. the heat radiated from a fire). Wind and air temperatures have no effect on this heat transfer.

Conduction: Heat is transferred in the interface between skin and material.

Evaporation: The conversion of a liquid to vapour requires heat. When sweat, or water, on the surface of the body evaporates, heat is lost, and the body cools. Given a sweat evaporation rate of 1 litre per hour, 680 Watts, (580 kcal/hour) are emitted, if all vaporisation has taken place on the surface of the skin.

During *respiration*, the air is moistened and heated. Due to inhaled air being cold and, consequently, very dry, heat is lost in both warming and moisturizing the inhaled air. This is especially so when we breathe through the mouth, because no use is made of heat exchange in the upper airways.

Heat exchange between the clothed surface of the body and its surrounding varies according to clothing, activity and climate, (wind and sun, for example). In the case of a person at rest or with a low level of activity, heat loss through respiration equals about 10 per cent of energy production (Fig 1). Convection normally accounts for 50 per cent or more of all heat emissions. Radiation accounts for less than 30 per cent. Conduction is negligible, except when sitting or lying on cold surfaces. Evaporation is a less suitable form of heat emission in cold weather and is normally less than 10 per cent. The reason for this is considered below, in the section headed Clothing. Convection and evaporation are the two forms with the greatest potential for increase.

Practical information

Use of special heat exchange breathing masks in cold weather leads to considerable recovery of both heat and moisture. By this means, respiratory heat losses can be reduced by 50 % or more.

Exposed, warm parts of the body, (face, head) lose a great deal of heat because of the big difference in temperature between the skin and the ambient air. A bare head with hair cut short can emit about 50 watts at

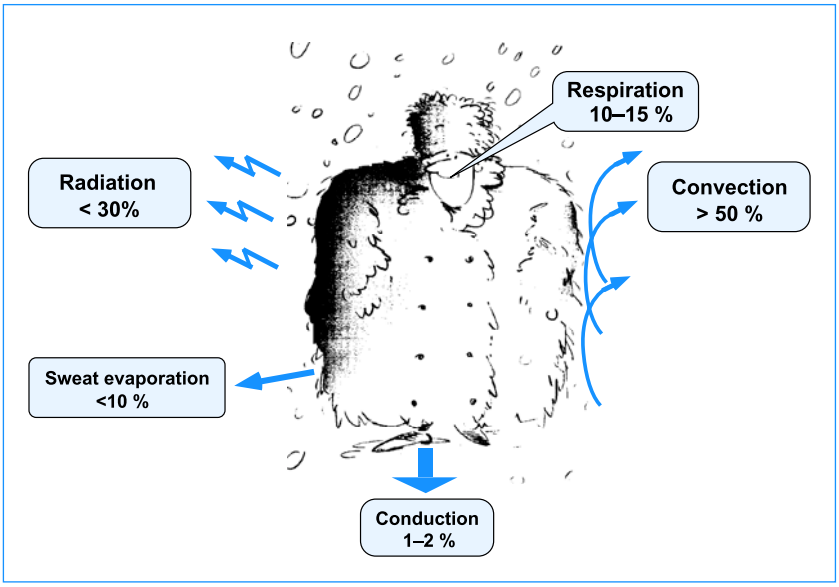


Fig 1. Approximate breakdown of body heat emission in cold weather.

-10 °C. For a person at rest and otherwise adequately dressed, this represents about 50 % of total heat emission.

During heavy exertion, when high perfusion of the skin is easily maintained, the bare hands can emit 100 watts or more, (-10 °C). If we are too warm, bare hands can be used as ”heat sinks”.

The temperature on the outside of thick clothing, at rest or during low activity, is only a few degrees higher than that of the surrounding air. For this reason measures to reduce heat radiation alone have limited effect (compare with the effect of a breathing mask).

Clothing

Most body heat, (more than 85 per cent) is emitted from the skin. In the majority of cases this also means that the heat will pass through one or more layers of clothing. This makes heat transport more complicated. In a simplified analysis, clothing can be regarded as a homogeneous layer resisting the flow of heat. The resistance can be measured and thus used for gauging the amount of heat loss. The resistance value, (or thermal insulation) applies to the whole clothing ensemble. It is stated in $m^2°C/W$, but a more popular expression is clo-value (1 clo = 1.155 m^2 °C/W. Clo-values are measured with the aid of a thermal manikin according to European standard ENV342. Some examples of clo-values will be found in Table 2.

Table 2. Examples of insulation values of complete clothing systems (static, wind-still values).

Clothing	clo	m^2 °C/W
Underpants, T-shirt, shorts, thin socks, sandals	0.30	0.050
Underpants, short-sleeved shirt, thin trousers, thin socks, shoes	0.50	0.080
Underpants, jogging vest + trousers, knee-length socks, trainers	0.75	0.120
Underpants, shirt, trousers, jacket, socks, shoes	1.00	0.155
Underpants, T-shirt, shirt, trousers, jacket, thermal jacket, socks, shoes	1.25	0.190
Underpants, T-shirt, shirt, trousers, jacket, thermal jacket + trousers, socks, shoes	1.55	0.225
Underpants, T-shirt, shirt, trousers, jacket, outer jacket with thick lining + dungarees, socks, shoes	1.85	0.285
Underpants, short-sleeved vest, shirt, trousers, jacket, outer jacket and trousers with lining, socks, shoes, hood, gloves	2.00	0.310
Long johns, long-sleeved shirt, thermal jacket and trousers, thickly lined jacket and outer trousers socks, shoes, hood, gloves	2.55	0.395
Long johns, long-sleeved shirt, fleece jacket and trousers, thickly lined parka and outer trousers, socks, shoes, hood, gloves	3.25	0.500
Arctic clothing systems	4–4.50	0.62–0.7
Sleeping bags	3–12	0.47–1.9

This table shows only the clo-value of clothing. Surrounding the actual clothing is a more or less untouched layer of air, which contributes additional insulation. During repose and in calm weather, this can be 0.8 clo. Wind and body movement can reduce it towards 0 clo. Thus a thinly dressed person loses a larger percentage of their insulation through movement and wind chill than a well dressed person does.

The effect of wind on the total thermal insulation of three types of winter clothing is illustrated in Figure 2. The clothing comprised underwear and an insulating middle layer. Three types of outer garments, (jacket and trousers) with different air permeability were used. The most impermeable, (wind-proof) ensemble reduces insulation by 40–50 % at 10–20 m/s. A porous outer layer causes reductions of 80–90 %. The typical wind-tight material of most winter wears cause reduction by 50–60 %. In practice this means that heat losses are almost doubled.

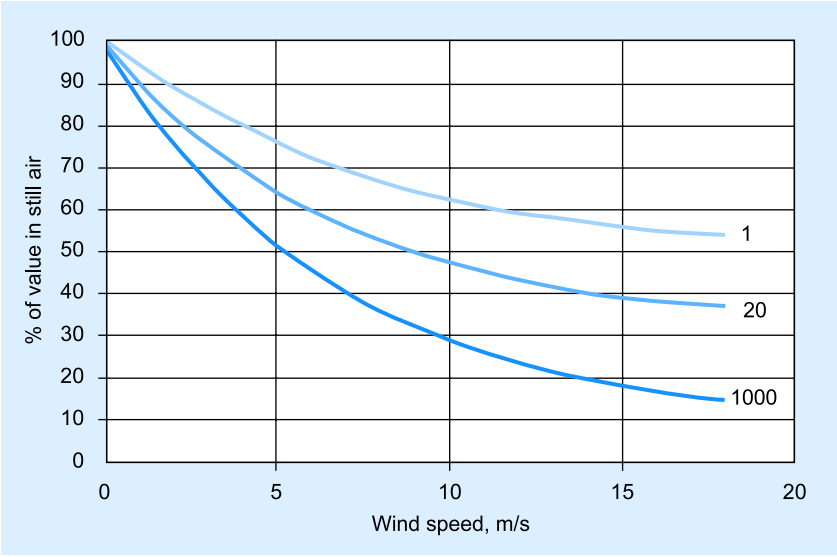


Fig 2. Effect of wind on thermal insulation. Remaining insulation compared to calm conditions are given for three levels of air permeability of the outer layers. Number 1 is highly wind-proof, 20 is wind-tight and 1000 is permeable to wind penetration.

The insulating capacity of clothing depends above all on its ability to retain a layer of stationary air. Insulating capacity, therefore, is on the whole directly proportional to the thickness of the layer of clothing, (1.3 clo per cm material). The fibre of a material makes less difference than its thickness.

Warm clothing means thick clothing. This creates problems in sweaty situations, because high insulation automatically means high vapour resistance, i.e. difficulty for the evaporated sweat to pass through. In a cold climate, temperature in the layers of clothing falls steeply, above all in the outer layers. Therefore most of the vapour condenses and possibly freezes in the outer layers, thereby releasing heat. This heat can be transported outwards, but moisture reduces the effectiveness of insulation. In situations offering limited opportunities for a change of clothing and for drying this can cause serious problems when the body's own heat production diminishes in connection with rest and overnight stays.

A certain amount of ventilation takes place inside clothing when we move. This helps to pump out heat and water vapour. Flexible clothing must provide good opportunities for ventilation in connection with strenuous activity.

One important rule of thumb for a cold climate is that modifications of clothing must precede or come at a very early stage of changes of activity, to counteract sweating.

Practical information

The difference in insulation between different materials of the same thickness is small and usually of no practical importance. The good properties of woollen materials can be ascribed to their thickness and also to the resilience of the woollen fibre and its capacity for retaining and recovering its thickness after compression. Wool also retains its resilience, given moderate moisture absorption.

Down also has a natural airiness which provides good insulation, but, when it becomes wet, it rapidly loses its ability to insulate. This loss of insulation is also true of cotton when it becomes wet.

Most synthetic fibres, suitably structured, can impart good insulating and moisture transfer properties to clothing. Synthetic materials, as a general rule, absorb only a little moisture. The moisture is therefore capillary-bound or condenses on the surface of the fibre. Given the same production of sweat, this usually means less moisture in the clothing and, consequently, faster drying.

As with all materials, these properties will be retained longer if the clothes are well looked after. Several extreme expeditions however, have shown that the possible deterioration is probably marginal, at least from the perspective of a few weeks' service. Some expeditions have travelled to the poles and climbed Everest wearing clothes of both natural and synthetic materials. Many synthetic materials have the advantage of high wear strength and low weight.

Heat reflective layers attached to clothing or sleeping bags do very little, if anything, to improve insulation. So-called space blankets of thin foil or felt provide wind protection (at moderate wind velocities) but no appreciable improvement of insulation. The same degree of wind protection and insulation can be obtained with a plastic bin liner, similarly used.

Wind-proof materials are necessary in cold and windy weather. They practically limit the effect of the wind to the outer layer of air. Clothing ought not to be compressible by strong wind.

Materials which "breathe", i.e. admit water vapour, are of limited usefulness in cold weather for reasons already given. For the most part water vapour does not get through to the outer layer, because both dew point and freezing point are often located inside the layers of the clothes. The vapour condenses into liquid and the liquid freezes to ice. This type of material can be of some advantage during rapid alternations between cold and a warmer climate.

Hands and feet are greatly dependent on the supply of heat carried by the bloodstream. There are no gloves or mittens capable of keeping hands and feet warm in severe cold when endogenous heat supply diminishes.

Behaviour

As mentioned earlier, the feasibility of a good thermal balance in cold conditions depends on an interaction between climate, clothing and behaviour. Good clothing and equipment are important components, but they cannot take the place of knowledge and experience. Farsighted, sensible and appropriate choices and adaptation of clothing, equipment and behaviour to varying circumstances are a vital component.

Sweating must as far as possible be avoided during prolonged exposure to cold. Flexible clothing and constant adaptation of clothing and activity are keys to good thermal control. Help is also obtainable in this respect by temporarily using the hands and head as ”heat sinks”.
Wind accelerates heat loss, even if one is wearing windproof outer garments.

Cold stress assessment

Problems and risks can be assessed with reference to different types of hypothermia.

1. General hypothermia
2. Local cooling
   - Extremity cooling
   - Wind chill
   - Contact cooling
   - Airway cooling
3. Hypothermia in water

General hypothermia

On the basis of the thermal balance equation outlined above and related expressions for calculating the different forms of heat emission, one can determine the insulation requirement which clothing has to meet in order to create good thermal balance under various conditions. A special method (IREQ; Insulation REQuired) has been developed for this purpose (ISO-TR11079). The insulation requirement (IREQ-value) can be calculated on the basis of air temperature, mean radiation temperature, air humidity and wind, together with the magnitude of the body’s heat production.

The IREQ-value is the insulation that the clothing worn has to provide, under the prevailing conditions, in order to create thermal equilibrium. For practical use of the IREQ method, one needs to know or find out the insulating capacity of the clothing that is or will be worn (see Table 2). A European standard for cold protective clothing describes a measuring

method for this purpose (ENV342). The insulation of the complete clothing ensemble is measured with a thermal manikin. Using the IREQ method, one can

- determine the insulation requirements in actual working situations (Fig 3)
- calculate appropriate maximum working time if the clothing worn does not cover the need (Fig 4)
- plan work inputs according to climatic conditions and the protective clothing available.

It is important to remember that IREQ method can only provide guidance. Final adjustment of clothing and working time has to be done by the individual, according to own needs and circumstances. Thermal insulation of clothing reduced by wind for example (Fig 2). Body movements increase ventilation of clothing, which may reduce insulation by 10–20 %.

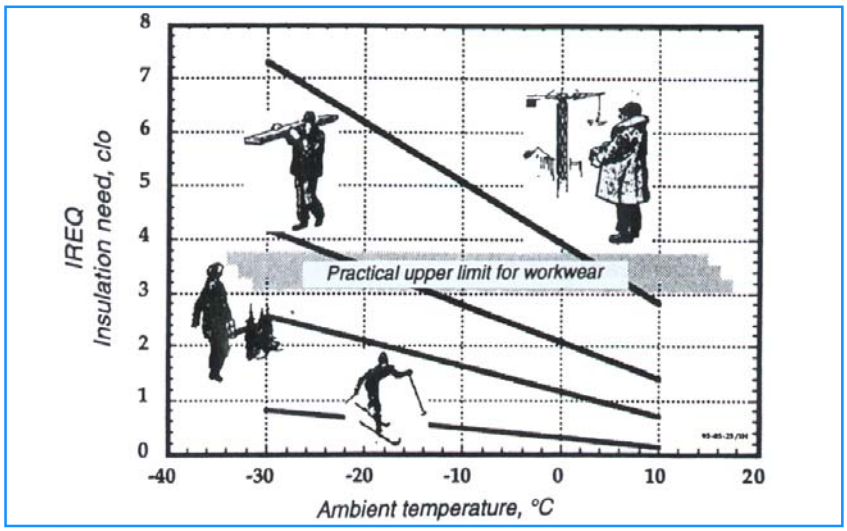


Fig 3. Clothing should provide this much insulation in order to achieve a good thermal balance. Very good wind-proof protective clothing provides, at most, 3–4 clo. If more is needed, working time has to be limited.

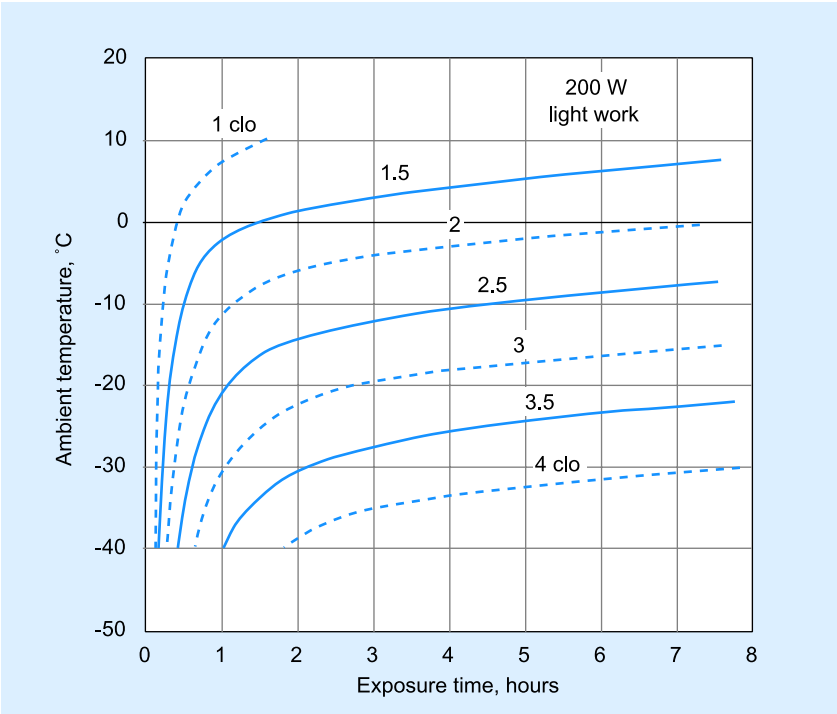


Fig 4. Calculated work time in different air temperatures for 7 different levels of clothing insulation (compare Table 2). The values refer to calm weather. Wind reduces the clo-values and requires additional clothing. Air permeability of the outer garment plays an important role (compare Fig 2). Air permeability for these garments' outer layer is 8 l/m²s.

Local cooling

The problem when working outdoors in the cold is often not about keeping the body warm but about avoiding different forms of local cooling. As mentioned earlier, a good thermal balance is important and helps to reduce, though it does not eliminate, the risk of local cold injury.

Extremity cooling

Capacity for keeping the hands and feet warm varies a great deal from one individual to another. It depends partly on knowledge, experience and behaviour, partly on physiological differences and, in certain cases, on medical disability. Hands and feet are extremely susceptible to cooling. As mentioned earlier, internal heat supply falls very drastically when the body starts to get cold. It is theoretically possible to calculate the combinations of temperature, wind, glove insulation and time that involve a risk of cold injury. The graph in Figure 5 is based on calculations of this kind.

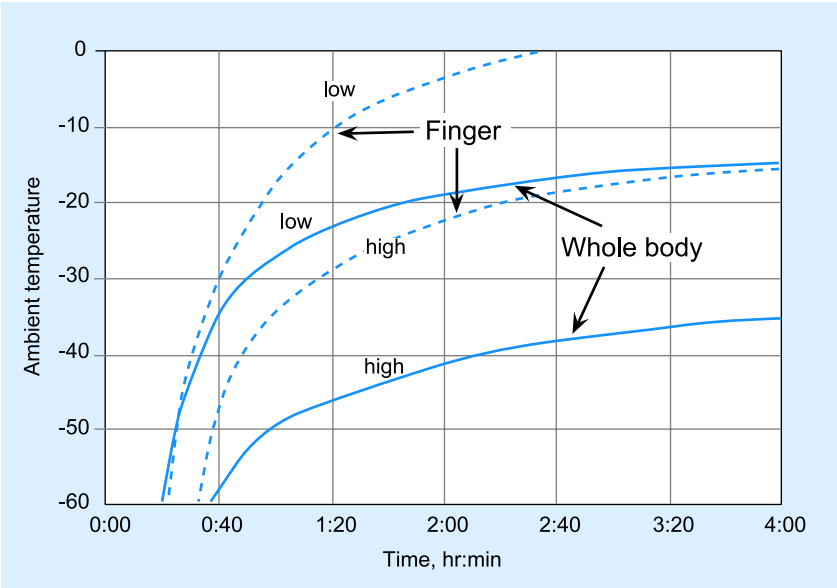


Fig 5. Time taken by fingertip temperature to fall to +5 °C at two comparative levels of activity. Clothing corresponds to 3 clo and gloves to 2 clo.

With poor circulation in the hands and feet, there is a serious risk of frostbite after only one hour at -20 °C. If a person is working and keeps his body warm (high activity), the fingers can still be kept warm below zero and for a couple of hours at -20 °C. The graphs in the figure also show that at very low temperatures the exposure times calculated for the whole body and for fingers coincide, (compare Fig 5).

Wind chill

Wind chill is a well-known phenomenon, especially in a cold climate. Table 3 can be found, in various formats, in many different contexts. They all have the same origin, namely measurements of the local cooling effect caused by the wind on *bare* skin. The cooling effect (heat loss) has then been linked with an assessment of the risk of cold injury (frostbite). Instead of indicating the heat loss at which different effects occur, the table gives the equivalent cold temperature. This corresponds to the actual temperature in calm weather, which has the same cooling effect on the skin as the combination of air temperature and wind referred to. This approach has an instructional value, because low temperatures transmit the ”right” warning signals to the user.

A wind speed of 16 m/s (60 km/hour) at a temperature of -10 °C has the same effect on bare skin as -34 °C in calm weather. Scooter and moped riding, ski joring, downhill skiing etc. can produce a rapid slipstream, which moreover is accentuated in a head wind.

A temperature below -30 °C implies a real risk of cold injury if exposure is prolonged (for hours), while a temperature below -60 °C can inflict cold injury in a minute or so.

This table is not to be used for assessing whole body hypothermia.

Table 3. Wind chill on bare skin as a function of wind speed and air temperature expressed in a so-called equivalent cold temperature (after ISO-TR11079).

Wind		Air temperature								
m/s	km/h	0	-5	-10	-15	-20	-25	-30	-35	-40
2	**7**	-1	-6	-11	-16	-21	-27	-32	-37	-42
5	**18**	-9	-15	-21	-28	-34	-40	-47	-53	-59
8	**30**	-13	-20	-27	-34	-41	-48	-55	-62	-69
16	**60**	-18	-26	-34	-42	-49	-57	-65	-73	-80
25	**90**	-20	-28	-36	-44	-52	-60	-69	-77	-85

This table is based on the original formula from Paul Siple 1945, but these values are based on factual wind-speeds compared to wind-speed intervals in the corresponding table in the main text. Editor.

Contact cooling

Tasks requiring a high level of precision and dexterity often have to be done with bare hands. Fingers and hands touching cold objects soon become cold themselves, especially if the objects are made of metal. This involves a serious risk of local pain, numbness and frostbite when the temperature is below freezing. Fingertip temperature when touching aluminium at -5 °C falls nearly to freezing point within a few seconds and gradually falls below freezing.

Liquids have high thermal conductivity, and so all liquids with a freezing point below 0 °C present a latent risk of cold injury when handled manually. This applies, for example, to fuels and solvents. Cold liquids spilt on an unprotected hand will swiftly produce a cold injury. Cold liquids, therefore, should never be handled with bare hands.

Bronchial cooling

Inhalation of cold air cools the airways. The colder it is and the harder one exerts oneself, (breathes) the greater and deeper the cooling effect will be. A certain heat exchange takes place in the airways, especially the upper ones, but this is not sufficient in extreme situations. Sportsmen often incur bronchial disorders when training and competing in the cold, and very often they develop a hypersensitivity reaction resembling asthma. Light and moderately heavy work entails less risk, but there is still cause, at temperatures below -30 °C, to protect the airways with some kind of breathing mask. Masks of various kinds with a heat exchange function are commercially available.

Hypothermia in water

Immersion in cold water presents a major risk of serious hypothermia. Water temperatures in seas, lakes and watercourses (in Sweden) for the greater part of the year are so low they can be directly life-threatening after only a few hours' immersion. Physical constitution, clothing and behaviour are important factors influencing survival time. Obesity, and especially thick subcutaneous fat, helps, as thin and diminutive persons, children especially, lose heat more rapidly.

Clothing which is not a direct impediment must be retained in the water. It is also important to keep still, with buoyancy support, until help arrives. Even proficient swimmers have a very limited capacity for swimming long distances in very cold water. The appropriate course of action will of course depend on the circumstances at the time of the accident, but the choice has to be made quickly, because even slight hypothermia reduces capacity for judgement and action.

The first few seconds in cold water easily provoke panic and desperate shortage of breath, (cold shock). If care is taken to overcome this, then even in extremely cold water, if one is fully dressed, it will take at least 30 minutes for the body to become seriously chilled.

Wetsuits, especially if insulated, permit longer immersion time. With insulated suits muscular exertion can help to reduce the rate of heat loss, i.e. quite contrary to the recommendations which have just been made.

Survival times can be calculated using equations similar to those underlying Figure 3. Conditions here are more complicated, however, due to the high thermal conductivity of water (25 times that of the air stratum surrounding the body). The chart in Figure 6 was produced with the aid of Wissler's computer model and is often used as a basis for assessments. The clo-values given refer to "wet" clo-value, i.e. protection

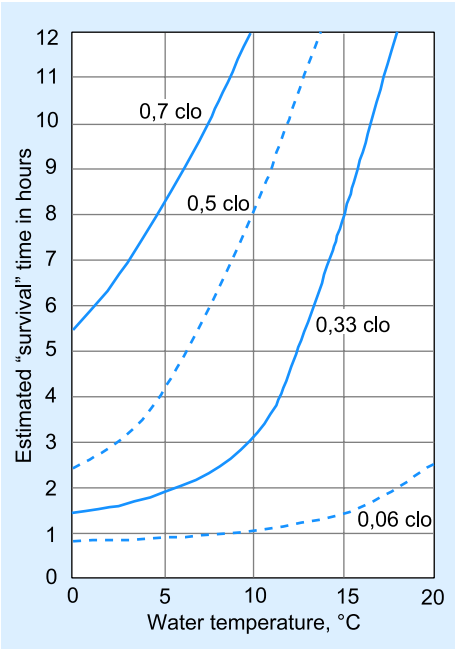


Fig 6. Estimated time taken by body temperature in still water to reach 34 °C for persons with little skin fold thickness (10th percentile). The clothing insulation values apply to the actual protection in water.

in the water, and probably correspond roughly to the span between light summer clothing and insulated wetsuits. The water temperature during the shipwreck of Estonia on 28 September 1994 was approximately 11 °C. Depending on the protection available, the estimated "survival" would be anything from less than an hour up to more than 6 hours. Several persons were rescued alive after more than 6 hours in the water.

How much cold can we tolerate?

One question which many people ask is, how cold can the atmosphere get before work becomes impossible? This is not an easy question to answer. Sustained work of low intensity and requiring manual precision already becomes difficult at about 0 °C. Heavy work requiring less precision can be done at temperatures below -20 °C. As has already been described, the maintenance of thermal balance is crucial and factors like workload, duration of work and quality of protection determine if work can continue. Important factors are also the nature and organisation of work (e.g. confinement to a particular workplace and duties, steady or variable working pace, opportunities for break and intermissions and a warm place to rest in), close contact with fellow-workers, vehicles and communications equipment, distance, terrain conditions, weather prospects etc.

Cold work – some practical hints

- Temperature loss in hands and feet, especially when activity is low, most often decides the lowest appropriate temperature and maximum working time.
- Wind chill temperatures below -30 °C should prompt stronger protection and a limit on the duration of work, while below -60 °C work should be suspended immediately.
- The insulation requirement for stationary, work is so high that few *practical* clothing systems on the market can provide continuous protection below about -10 °C.
- If protection is insufficient, working time must be reduced according to the clothing insulation actually available (cold protective clothing should be tested according to **ENV342**).
- Airway protection should be used at temperatures below -30 °C.
- Metals or liquid colder than 0 °C must not be touched with the bare hands.

Often, when the weather turns really cold, there is no point in doing any work, in view of the low level of efficiency and productivity. But there are some emergency situations when work has to be done, nevertheless.

Situations of this kind can be assessed on the basis of the following table. One necessary precondition for applying the figures in Table 4 is access to the best possible protective equipment (of course!). Every new spell of work must be preceded by full recovery in a heated space. The times are only for guidance and can never be forced on the individual. Because tolerance and adaptability vary so greatly from one individual to another, each person must be at liberty to stop work when they finds the situation dangerous.

Table 4. Recommended exposure times for light and heavy work respectively (see preconditions in the main text).

Air temperature	Work intensity Low	High
-30 °C	40 min	90 min
-40 °C	30 min	45 min
-55 °C	<20 min	<30 min

How are the problems tackled?

All activities become more complicated and more demanding and proceed more slowly in a cold than in a temperate climate. Clearly these conditions must already be taken into account when work is planned and conducted. Other actions should be aimed at assuring the individual of a good thermal balance and generally doing everything possible to facilitate the work in hand.

Some practical examples:

- Take difficulties and problems into account when designing, planning and scheduling activities.
- Adapt the working pace, the mode of operations, clothing and working time to prevailing conditions.
- Have a change of warm clothing available and change at least once a day.
- "Ventilate" and dry clothing and footwear, especially during long rewarming breaks.
- Check that the equipment supplied is adequate and appropriate.
- Alternate between activity and breaks of suitable length in a heated space.
- Avoid smoking, especially during short breaks, because it impedes rewarming, especially of the hands and feet.
- Shield work sites and rest facilities from the wind and make use of sunlight or extra heaters.

- Keep tools and working materials warm.
- Keep an eye on your fellow-workers, ("the buddy system") and watch out for signs of hypothermia or frostbite.
- Make sure that first aid equipment, extra warm garments and windbreaks are readily available.
- Keep the head warm!

Knowledge and experience are a cure for many problems. When deciding between safety requirements and functional requirements, protection from the cold must come first. Only the individual himself can decide when his protection is adequate, and he must therefore be given the liberty to adjust his work load, which circumstances require. This is also an important prerequisite for good efficiency and high work capacity.

Further reading

Holmér I., Granberg PO, Dahlström G. Cold. Encyclopaedia of Occupational Health. Stellman ed. ILO. Geneva, 42.29-42.43, 1997.

Holmér, I. Col Indices and Standards. Encyclopaedia of Occupational Health. Stellman ed. ILO. Geneva, 42.48–58, 1997.

Holmér I, Kuklane K. Problems with cold work. Arbete och Hälsa 18, 255 pp. Stockholm, 1998, ISBN 91-7045-483-3.

Holmér, I. Risk assessment in cold environment. Barents Newsletter on Occup. Health and Safety. **1**: 77–79, 1998.

ENV-342. Protective clothing – Ensembles for protection against cold. 15 pp. 1998.

ISO/TR-11079. Evaluation of cold environments – Determination of required clothing insulation (IREQ). 31 pp. 1993.

Avalanche accidents

For reasons of terrain and climate, avalanche deaths are not as common in Sweden as in other areas of Europe, especially the France, Swiss, and Austrian Alps. The effect of an avalanche, however, is the same whether the accident occurs in Sweden, the Alps or the Himalayas.

The following section is the latest information available, based on wide-reaching experience and emphasises the need for rapid rescue and resuscitation if lives are to be saved. The majority of deaths are due to asphyxiation, not to trauma or hypothermia. The principles of management discussed below should be clearly understood by those likely to rescue and treat avalanche victims.

Editor

Dr *Bruno Durrer*, Lauterbrunnen, Switzerland and Dr Hermann Brugger, Bruneck, Italy.

Introduction

Over the last twenty years, 150 persons have been killed by avalanches every year in Europe and in the USA . (Fig 1). In the Alps, the winter of 1999 was especially dramatic with almost 150 deaths.

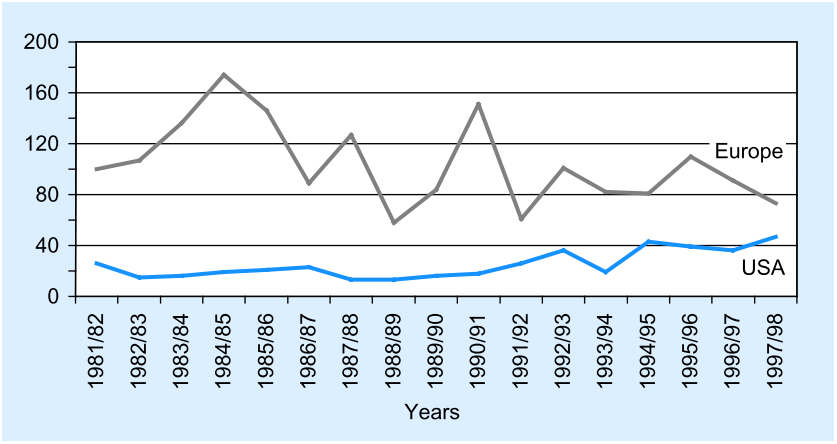


Fig 1. 1981–1998: Deaths by avalanches in Europe and in the USA.

The main cause of death in an avalanche is asphyxia. New data suggests that hypercapnia is an important factor also. Hypothermia and injuries are less important.

Causes of death in avalanches:

1. Acute Asphyxia (without air pocket) ca. 65 %
2. Prolonged Asphyxia with hypothermia (closed air pocket) ca. 25 %
3. Lethal injuries ca. 8–10 %

New data on avalanche survival and new diagnostic measures have changed the assessment of avalanche patients in the past few years.

Recent advances in Avalanche survival

Using the data of all avalanche victims in Switzerland from 1950 to 1974 (N: 481), the survival chances were calculated in the late seventies in relation to the time of burial. At the moment the avalanche stopped, the chances were 80 %. After one hour they decreased to 40 %, after two hours to 20 %, after three hours to 10 % and after 4 hours to 5 % etc. See Figure 2.

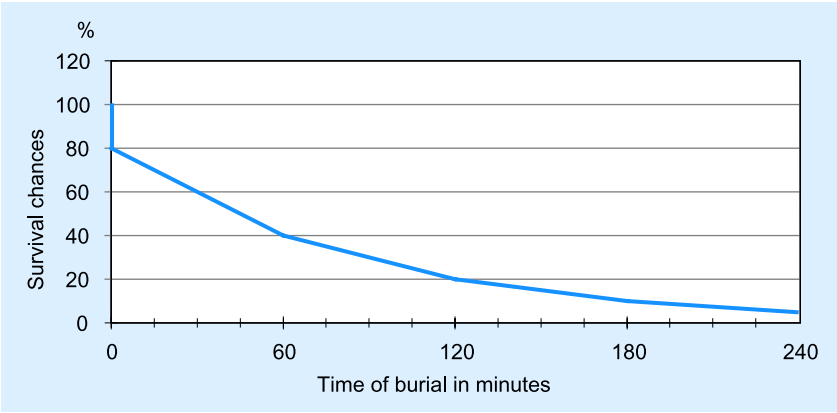


Fig 2. Earlier survival chances 1950–1974 (Schild et al.).

Avalanche rescuers however considered these survival chances as too optimistic and Swiss data from 1981 to 1991 (N: 933 skiers with 422 persons completely buried) and 1992–1998 (N: 638 skiers, snowboarders + 97 victims buried in buildings or cars) was calculated again with far greater accuracy. (Fig 3.)

Fig 3. Survival chances in the open field and in buildings or cars/trains (Falk, Brugger, EISLF)(Swiss Institute for Snow and Avalanche Research: Eidg.Inst. Schnee- und Lawinenforschung) Total completely buried 1981–1998, black curve. Completely buried in buildings (car/trains) 1981–1998, blue curve. Completely buried in the open field 1981–1991, black dotted curve.

Survival after an avalanche can be divided into four phases

1. Survival phase
2. Asphyxia phase
3. Survival only if an air pocket is present
4. Rescue

In the new curve, the initial survival probability is much higher then previously assumed. Only 8 % of skiers die within the first 15 minutes, mainly due to fatal injuries. The survival probability then drops from 92 % at 15 min to only 30 % at 35 min due to asphyxia of all victims without an air pocket. Each cavity in front of the mouth and nose, even if it seems to be very small and confined, is to be declared an "air pocket". The statement "no air pocket" can be made only if mouth and nose of the patient are blocked airtight by avalanche snow (or debris).

The constant survival chances between 35 and 90 min indicate a minimal risk of dying for buried persons with an air pocket. It is known that the snow cover prevents rapid cooling (maximally 3 °C per hour) and that oxygen consumption decreases significantly with lowering of the body temperature and loss of consciousness. The survival probability then falls from 27 % at 90 min to only 3 % at 130 min. There is an increased risk during the excavation due to a possible destruction of the air pocket and due to the possible after-drop with cardiac arrest. The survival chances of buried persons in buildings or cars/trains (n:97) are better than of persons buried in the open field. After 190 minutes the chances are still over 30 %.

The longest, documented survival times of completely buried avalanche victims were in an open slope (44 hours, Italy 1972) and in a building. (13 days, Austria 1951).

Practical consequences for the rescuers

The speed of extrication and the existence of an air pocket are the two most important factors for survival. Reduction of the present high mortality rate depends on increasing the proportion of victims freed within 15 min, which realistically means by first responders.

The new time goal for professional help (organized rescue) is extrication within 90 minutes. In buildings the survival chances after 190 minutes are higher than in the open slopes.

Practical consequences for mountaineering organisations

All ski-mountaineers have to be trained in safety precautions, as well as the basic techniques of searching, extracting and resuscitating avalanche

victims. At present many skiers carrying rescue transceivers are insufficiently familiar with their use, with fatal consequences.

The "avalanche air bag", possibly in combination with the "avalung", (jacket with mouthpiece, allows to move expired air to the back and avoids Carbon dioxide poisoning) might be the safety device of the future, but prophylaxis of avalanche accidents will always remain the only reliable safeguard.

Practical aspects of the medical treatment on site of hypothermia and avalanche victims

For practical rescue work and especially for the instruction of non-medical rescue staff, five stages of hypothermia should be distinguished. Staging criteria are the degree of consciousness, the presence or absence of shivering, the cardiac activity and the core temperature.

(This scoring system is slightly different from that in the main text, but its meaning is clear. Editor.)

HT I:	Clear consciousness with shivering	
	Core temperature	35–32 °C
HT II:	Impaired consciousness without shivering	
		32–28 °C
HT III:	Unconsciousness	
		28–24 °C
HT IV:	Apparent death	
		24–13,7 ? °C
HT V:	Death due to irreversible Hypothermia	
		< 13,7? (< 9 ?) °C

Reversible or irreversible hypothermia ?

In cases of severe hypothermia with cardiac arrest, non-medical personnel are not allowed to diagnose death at the accident site.

Prior to establishing death in the field, the emergency doctor has to exclude severe hypothermia. An ECG and a field thermometer are needed to make this diagnosis. For patients with cardiac action, the tympanic temperature measurement is recommended and for patients with asystole the oesophageal measurement is preferred. Resuscitation for the wrong reasons can expose the rescue team to unnecessary risks.

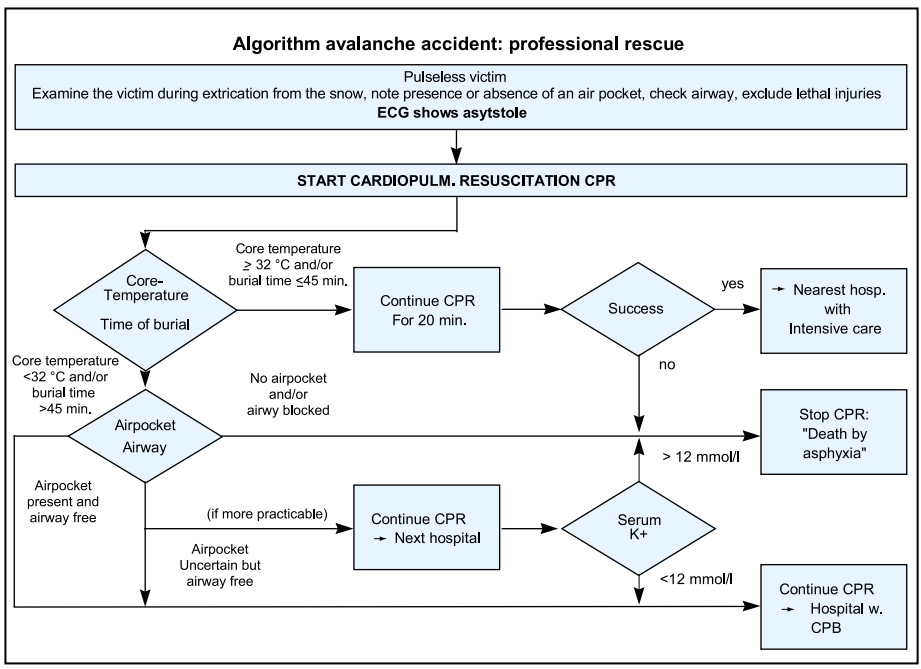
To date, the lowest documented reversible core temperature has been 13,7 °C in accidental hypothermia and 9 °C in induced hypothermia.

Recent data permits the diagnosis of death in avalanche victims if the serum potassium level is above 12mmol/L. More information is needed about the upper reversible limits of serum potassium (due to haemolysis, rhabdomyolysis) and about measuring devices which can be used in the field.

Recently, in some clinical centres even severe trauma patients were rewarmed by cardiopulmonary bypass (CPB). Consequently, it has to be decided on-site whether the patient is severely hypothermic with additional injuries or has died from injuries and subsequently cooled down.

Avalanche victims with asystole: Death by asphyxia or reversible servere hypothermia?

Algorithm 1



The successful use of CPB (Cardio-pulmonary Bypass) in hypothermia treatment led in the 1980's to no avalanche victim without fatal injuries being declared dead before clinical rewarming. The outcome in asystolic avalanche victims after CPB was disappointing, because in most buried victims cardiac arrest was caused by asphyxia and not by hypothermia. For the triage of asystolic avalanche victims (not fatally injured), the differential diagnosis between asphyxia and severe hypothermia plays a central role. The diagnostic criteria used in the algorithm are based on the

latest information on the pathophysiology of hypothermia in burial by avalanches:

In all those buried in avalanches without an air pocket, death occurs through acute asphyxia, within 45 minutes after burial. After this time, the existence of an air pocket forms the main criterion for triage.

In hypothermic patients the danger of circulatory instability begins below a core temperature of 32 °C. This temperature is achieved by those buried in avalanches, as a rule after 90 minutes, but not before 45 minutes, based on an average rate of cooling of 3 °Celsius per hour.

The medical on-site treatment of avalanche victims

These recommendations, approved by the Medical Commission for Alpine Emergency Medicine (ICAR), apply to the European Alps with many rescue stations and short flight distances to hospitals. In other regions these recommendations have to be adapted to the local rescue systems and to the local medical facilities.

On-site treatment of serve hypothermia in asystole (HT IV)

In case of severe hypothermia with asystole, cardiopulmonary resuscitation (CPR) is started (including intubation and ventilation preferably with humidified warm oxygen) as soon as its continued maintenance can be guaranteed. The frequency of external heart massage is the same as in normothermic patients.

Whether or not an apparently dead patient (stage HT IV) should be prevented from further cooling is still controversial (metabolic icebox vs. irreversible lower limits of the core temperature). During the excavation and evacuation of a patient, the core temperature may drop beyond reversible limits. For this reason most rescue doctors consider that proper protection against further cooling of an apparently dead patient (HT IV) is necessary. This is usually achieved by insulation and heat packs on the trunk. I.V. medication and perfusions are not considered to be mandatory in hypothermia with asystole. The air-transport of the victim to a hospital with CPB (Cardio Pulmonary Bypass) facilities is recommended.

On-site treatment of severe hypothermia in ventricular fibrillation

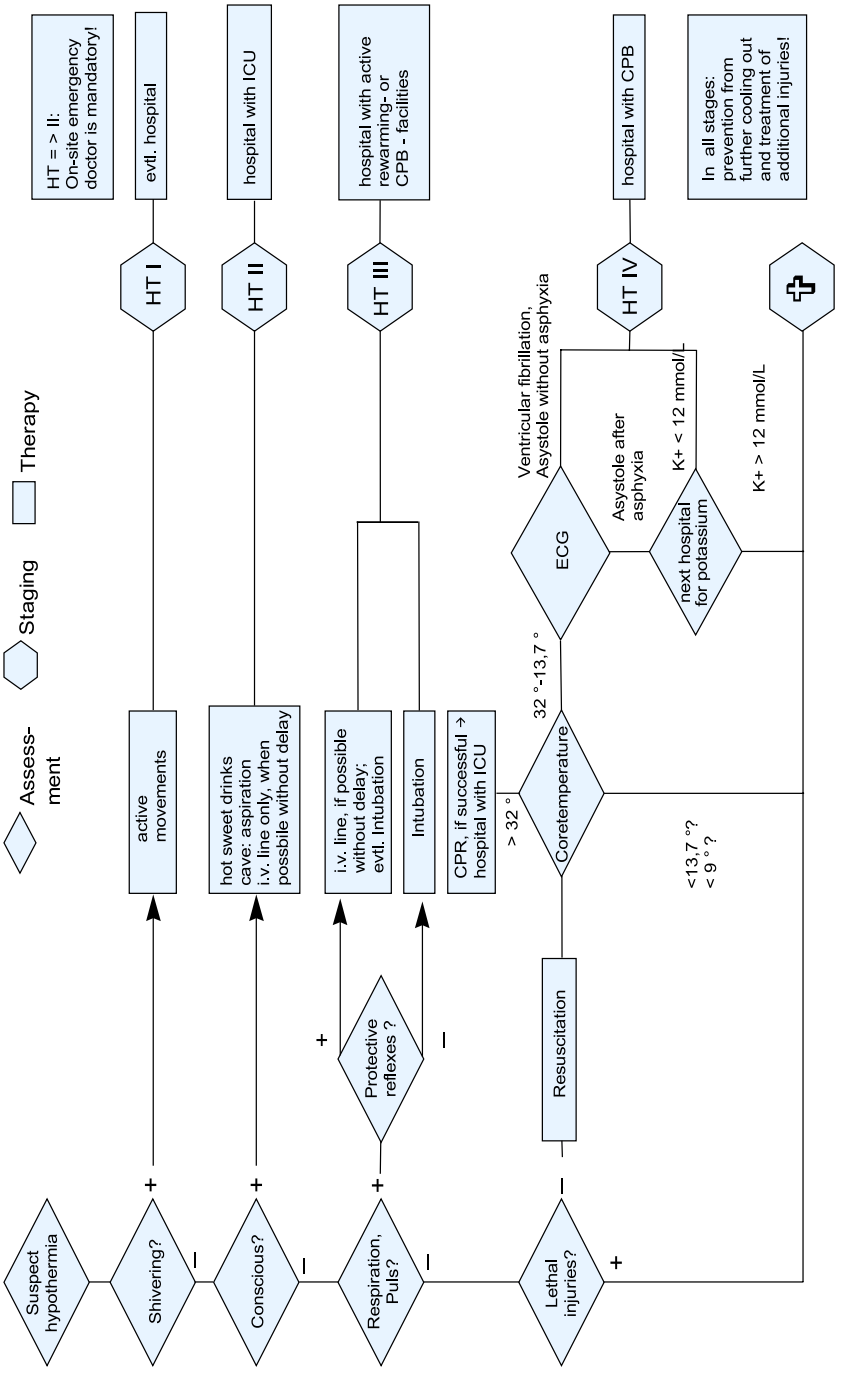
The on-site treatment is the same as in victims with asystole. Defibrillation below 30 degrees core temperature is supposed to be ineffective.

On-site treatment of servere hypothermia with pulse

Unconscious victims: HT III

Very careful handling avoids life-threatening arrhythmias. Whether it is absolutely necessary to intubate the unconscious patient at the site of

Algorithm 2. On-site treatment of hypothermia



accident is still a matter of discussion. In the Alps a patient is evacuated by helicopter within 15 to 20 minutes to a medical centre and the risk of a further heat loss during the treatment and transport has to be evaluated in relation to the advantages of intubation. For intubating a patient still with protective reflexes, an intravenous line is needed for the administration of medications. In unconscious patients (HT III) peripheral vessels are difficult to locate and establishing an intravenous line can be very time consuming. Since there is always an increased risk for further cooling in HT III, proper protection against further heat loss (e.g. airway warming) is of outmost importance. ECG monitoring has to be started as soon as possible. Transport of the victim to a hospital with CPB facilities is recommended.

At the moment little data exists about the danger zone for triggering arrhythmias in hypothermia. Between core temperatures of 32 °C to 30 °C, the risk of ventricular fibrillation is considered to be very low. Below 30 ° and especially below 28 °Celsius, the risk of arrhythmias increases. However, for establishing algorithms, the limits have to be on the safe side – always in favour of the patient.

Conscious victims: HT II / HT I

In a victim with impaired consciousness, very careful handling is necessary to avoid initiating life-threatening arrhythmias. If swallowing is possible, the administration of hot and sugared drinks is recommended. Strict control of the vital functions is necessary. The victim should be transported to a hospital with an intensive care unit.

Due to altitude and wind exposure in the mountains, hypothermia often occurs in combination with injuries. Airway warming, changing wet clothes for dry, hot drinks and insulation help to prevent further cooling out of the patient. Uninjured victims with shivering (HT I) do not have to be transported to a hospital in all cases.

Further reading

Brugger H. & Falk M.: Neue Perspektiven zur Lawinenverschüttung. Wien. Klin. Wochenschr. 104/6: 167–173 (1992).

Brugger H.& Falk M.: Die Phasen der Lawinenverschüttung. Notfallmedizin 19: 22–27 (1993).

Brugger H., Durrer B., Lawinenverschüttete mit Asystolie: Triage durch den Notarzt, Jahrbuch 1994 der Oesterreichischen Gesellschaft für Alpin und Höhenmedizin

Brugger H, Durrer B., Falk M., Notfallmedizinische Massnahmen bei der Lawinenverschüttung in Abhängigkeit von der Verschüttungsdauer,

Jahrbuch 1994 der Oesterreichischen Gesellschaft für Alpin und Höhenmedizin.
Brugger H., Durrer B. Adler L., On-site triage of avalanche victims with asystole by the emergency doctor, Resuscitation 31 (1996) p.11–16.
Danzl Daniel F et al., Accidental Hypothermia in Wilderness Medicine, C.V.Mosby Company, St.Louis, 1995.
Dubas F., Henzelin R., Michelet J. in: Colour Atlas ofMountain Medicine, Wolfe Publishing, London: 108–109 (1991).
Durrer B.: Allgemeine Unterkühlung: Messung der Kerntemperatur als Beurteilungshilfe. Rundbrief der österreichischen Gesellschaft für Alpin- und Höhenmedizin, Kaprun (1990).
Durrer B.: Hypothermie im Gebirge: Ärztliche Maßnahmen am Unfallort. Österreichisches Journal für Sportmedizin 2: 50–54 (1991).
Durrer B., Brugger H., Akzidentelle Hypothermie im Gebirge, Möglichkeiten und Grenzen der ärztlichen Erstversorgung, Jahrbuch 1994 der Oesterreichischen Gesellschaft für Alpin und Höhenmedizin.
Ennemoser O. et al.: Tympanonthermometer zur Messung der Körperkerntemperatur. ThermoMed 7: 63-65 (1991).
Falk M., Brugger H, Adler L: Avalanche survival chances, Nature Vol 368, p 21 (1994).
Flora G: Allgemeine Unterkühlung – örtliche Erfrierung. Zeitschrift für Allgemeinmedizin 28: 1503–1506 (1982).
Flora, G.: Alpine Unfälle und Bergrettungsdienst. Med. Welt 5: 223–228 (1964).
Kornberger E., Posch G., Koller J.: Die Wertigkeit der Körperkerntemperaturmessung beim Lawinenunfall und ihre technischen Probleme. Int. Bergrettungsärztetagung Innsbruck (1989).
Lloyd Evan L, Hypothermia and Cold Stress, Croom Hel m, London, 1986.
Locher Th., Walpoth B., Pfluger D., Althaus U.: Akzidentelle Hypothermie in der Schweiz (1980–1987) – Kasuistik und prognostische Faktoren. Schweiz. med. Wschr. 121: 1020–1028 (1991).

Durrer* Bruno, M.D., Mountain Guide, Swiss Alpine Club Rescue /Air Glaciers, CH-3822 Lauterbrunnen, Switzerland.
Brugger* Hermann, M.D., Mountain rescue service at the South Tyrol Alpine Association, Europastrasse 17, I-39031 Bruneck, South Tyrol, Italy.

*Medcom ICAR (Int.Comm. Alpine rescue)

The Anaris disaster

Dr *Börje Renström*, formerly Senior Consultant of Anaesthesiology, Östersund Hospital, Sweden. († 2001)

On 24th February 1978, six healthy young skiers set out from a chalet in the Lunndörr Pass in western Jämtland on a 14 km crossing of the Anaris Mountain to the next mountain chalet. They were copiously equipped. In addition to their daily ration of sandwiches and rose hip soup, for example, they also had emergency rations and had rented two mountain rescue packages containing wind bags, radio equipment and spades.

The first stage was an uphill climb of 2 or 3 km over a mountain ridge, and nearly all the skiers began to sweat. The temperature was -16 °C when they set out, with a very low wind of 5–6 m/s, but increasing all the time, and local cold injuries began to appear, as well as fatigue. When the weather deteriorated further, with ground snow drifts and a wind velocity of 20 m/s for example, the group decided to seek shelter. They dug a "ditch" in the snow, but because the position near the trail was windy, the shelter was only 8 dm deep and the roof of snow blocks showed signs of getting blown off.

Another three persons now joined the group. Altogether, they now had four wind bags as well as sleeping bags, but these were not used. When the person who was to be the sole survivor tried, unsuccessfully, to repair the roof of the snow bivvy, he did not re-enter. Because the snow had blocked the entrance, he kept moving and had something to eat. Neither he nor two of the new arrivals were hungry or felt very cold. When they began to understand that they were going to freeze to death, two of the party decided to hasten the process and walked straight out into the storm.

On the second day after the beginning of the accident, the sole survivor tried, with his bare hands, (because his mittens had blown away and frozen hands cannot hold a spade) to dig the others out, but without success. He did manage, however, despite frozen hands and feet, to get back to the chalet 4 km away, where he met a couple of people who contacted the mountain rescue service.

Only one of the nine survived, though with deep cold injuries to his hands and feet.

Two others who were thought by the mountain rescue police to have shown signs of life were taken by helicopter to hospital in Östersund, together with the survivor.

Lessons: The mountain radio did not work in the cold. The decision to take shelter was made late and poorly implemented. The terrain should have been inspected more carefully in the search for shelter: 15 or 20 metres to one side, the snow was 5 or 6 metres deep!

Equipment that stays in rucksacks because they cannot be opened with frozen hands is worthless. Mittens must be attached to clothing.

The clothing was copious, but the forces of nature were greater. Example: stout shoes, two pairs of woollen stockings, leggings, windproof trousers, skiing trousers, two pairs of long johns, weather-proof blouse, anorak with hood, woollen blouse, sports shirt, pullover, mittens, beanie. Trousers and stockings were saturated with urine. The need to urinate because of cold-diuresis could not be dealt with frozen hands: clothing was wet through right down to the shoes.

Hypothermic persons must not be carried sitting in a helicopter.

Resuscitation attempts in hospital should begin with a warm IV drip.

In CPR, bear in mind that slow-flowing viscous blood, which is concentrated by dehydration, needs time to enter the heart between compressions. With low metabolism, excessively rapid ventilation causes rapid alkalosis, with possible impaired oxygenation of the heart, and tetany. Advanced CPR is technically feasible during rewarming in a bath except for defibrillation.

The thesis "no one is dead until they are warm and dead" ought reasonably to be accompanied by "some victims are clearly dead when they are cold and dead" (Auerbach). After several hours' attempted rewarming in a bath of 40–42 °C, CPR, and warm drip at +41 °C, the thermometer did not rise above its lowest reading in this case, +19 °C. Rectal palpation revealed that ice was still present. Pathological examination showed among other things that all the deceased were oedematose, with multiple ulceration of the stomach, and all had wet clothing.

District Medical Officer Samuel Naucler's description of the clinical picture and treatment of hypothermia

From Proceedings of the Royal Swedish Academy of Sciences (1756).

An early Swedish description (1756) of hypothermia and its treatment is to be found in District Medical Officer Samuel Naucler's narrative "Of a man who, being apparently frozen to death, was helped to life."

Briefly, the narrative describes how, on 23rd March 1756, during a storm at sea, a man aged about 60 was washed ashore on a rock on the coast of Gotland. He was taken for dead and carried into a cottage. His feet were frostbitten, his toes were black. His legs and arms, stomach, chest and face were ice-cold. His joints were stiff, his eyes were wide open and he did not react to the touch. No breathing was apparent and no heartbeat was audible. Some little warmth could still be felt in the cardiac region, however, and so Naucler decided to try and "restore the lost circulation and lively motion of the blood". Alternating between the application of warmth and cold cloths, rubbing and "Torningen's balsam", Naucler was eventually able, after five-and-a-half hours' work, to feel the man's pulse. After another hour he was able, using a silver spoon, to part the man's jaws.

He now heated a little wine, adding to it 20 drops of the medicament "gratia probatum", which he poured into the man. As soon as it entered his mouth, the man bellowed like an ox and people thought he was giving up the ghost, but on the contrary, after a little resistance he swallowed both wine and drops. The narrative goes on to describe how the man became increasingly conscious and, towards the following evening, after eating "oat soup" and having a "good opening", happily returned home (Naucler 1756).