# ACSM Expert Consensus Statement: Injury Prevention and Exercise Performance duringCold-Weather Exercise

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## Abstract

Cold injury can result from exercising at low temperatures and can impair exercise performance or cause lifelong debility or death. This consensusstatement provides up-to-date information on the pathogenesis, nature, impacts, prevention, and treatment of the most common cold injuries.

## Introduction

American College of Sports Medicine (ACSM) Expert Consensus Statements are created by a small group of recognized leaders in a field. They highlight knowledge gaps, present existing knowledge, and provide recommendations for clinical practice. The new, shorter format is intended to makethe text more focused and accessible.

This statement addresses the deleterious aspects of expo- sure to cold. The use of cold in injury prevention, treatment, or recovery is beyond its scope and is dealt with elsewhere (1). As well, the reader is referred to review articles on the de-tailed physiological responses to acute and chronic cold expo-sure and effects of cold acclimatization (2,3). This statementupdates and replaces the ACSM position statement publishedin 2006, entitled “Prevention of Cold Injuries during Exer- cise” (4). As an official pronouncement of the college, it re- flects the college's position on the scientific and clinical aspectsof cold injury during exercise.

Many people work or exercise in or near a cold environment, be that cold air or cold water. Cooling can impair performance and threaten life, and coldis a leading cause of death among peopleengaged in sports (5). The breadth and seriousness of the challenge represented
by cold are reflected in the topics covered in this statement and include frostbite, nonfreezing cold injury (NFCI), hypo-thermia, avalanche burial, snow blindness, drowning, and sudden cardiac death. In addition, cold can constitute part ofa combined environmental threat, for example in combinationwith hypoxia in high mountains. It follows that an under- standing of the impact of cold environments and approachesto mitigate these threats is essential for those hoping to per- form in cold conditions.

## Cold Air (Frostbite)

Frostbite is a direct freezing injury occurring when the skinsurface freezes in saltwater at ~ −0.55°C (31°F) (6) and in airbelow −3°C (26.6°F) (7–9). Exposed tissues with poor perfu-sion are most commonly affected (hands, feet, head) (10–12).Exposure times for injury vary from seconds to hours, depend-ing on the type and intensity of cold exposure, degree of phys-ical activity, protective clothing, and various individual factors(Table 1) (47). At subfreezing tissue temperatures, extracellularice crystals form in susceptible tissue, leading to cellular mechani- cal damage and increased osmotic pressure, causing inflammation, microvascular thrombosis, ischemia, and hypoxia. Formation ofintracellular ice crystals may then occur. Thawing increases tissueedema and provokes an inflammatory response and reperfusioninjury (11). Frostbite is classified as described in Table 2.

Skin numbness is a sign of a heightened cold injury risk. Apale spot on the skin indicates superficial cold injury, which is characterized by partial skin freezing and mild edema (50).The injured area should be rewarmed by contact with warm skin (their own, or someone else's) and further cooling avoided.With more severe frostbite, the injured area is cold to the touch,and patients often complain that it feels “like a block of wood”(11). If possible, the frozen part or area should not be rewarmedunless refreezing can be avoided (51).

**Table 1.**
Factors predisposing to frostbite and NFCI.

**Climate**
Wind
Wetness, Immersion in Cold WaterContact with Cold Materials
High AltitudeHypoxia
Long Duration or High Amount of Cold Exposure

**Individual Characteristics and Physiology**
African-American or Afro-Caribbean Ethnic BackgroundMale
Children
Elderly
Previous Cold InjuryPoor CIVD Response
Homeless

**Behavioral**
Smoking Alcohol use
\Drug use
Inappropriate or constrictive clothing Prolonged stationary situation, immobility
Fatigue, dehydration, malnutrition
Use of emollients
Military rank/task
Poor calorie intake

**Individual/clinical**
Coronary artery disease/ischemic heart disease, cardiac insufficiency, stroke
Peripheral vascular disease
Peripheral neuropathy
Cold sensitivity
Raynaud's phenomenon, white fingers Hand-arm-vibration syndrome, vibration
Diabetes
Hypothyroidism, hypopituitarism
Depression, schizophrenia, dementia
Neurovascular diseases
Sweating or hyperhidrosis Previous cold injury
Medication (vasoconstrictors)

Frostbite occurrence ranges from 7% to 11% among thegeneral population in Scandinavian countries (10,13,14,52).It also occurs in military training (15,53,54). It is more com-mon in rural, northern climates (13,16), in occupations in-volving high physical strain and extended cold exposure(14); and in leisure/sporting activities, such as mountaineering(17,18,55,56), cold climate hiking (57), use of all-terrain vehicles in the cold or of snowmobiles (16); and sports activitiesgenerating high wind speed, such as alpine skiing or sledding(58), or associated with prolonged stationary posture, suchas kite skiing and hang gliding (59,60). A recent study concluded that the incidence of frostbite injuries in the AustrianAlps is low (56), mainly due to better awareness and clothing.Age (19), sex (10,13,14,16,50,56), and ethnic background (20,21) affect frostbite risk (Table 1). Diseases affecting neural,vascular, and metabolic functions and related tissue perfusionand microvascular function, as well as metabolic heat production also may increase frostbite risk. Autonomic and peripheralneuropathies (*e.g.*, diabetes) impair neural control and thermalsensations (22), central neurological disease (*e.g.*, multiple sclerosis, spinal cord injury) can impair mobility, thermoregulation, cardiac and vascular control; and vascular disease can impair tissue perfusion and responsiveness.

**Table 2.**
Traditional historical classification of frostbite adapted from Fudge (48).

| Frostbite Degree | Physical Findings |
|---|---|
| 1st Degree | Numbness, central white or yellow, waxy discoloration, surrounded by erythemaand edema, desquamation, dysesthesia |
| 2nd Degree | Surface blisters containing clear or opalescent fluid surrounded by erythema and edema |
| 3rd Degree | May initially present as 2nd degree, but hemorrhagic blisters appear within 24 h,tissue loss involving entire thickness of skin |
| 4th Degree | Injury is through the dermis, into subcutaneous tissue, muscle, and bone |

For field use, a simpler two-tier classification may be more appropriate (superficial — no or minimal anticipated tissue loss, corresponding to 1st- and 2nd-degree injury: deep — anticipated tissue loss, corresponding to 3rd- and 4th-degree injury (49).

Endocrine conditions(*e.g.*, hypothyroidism, hypopituitarism, adrenal insufficiency)can decrease metabolic heat production in the cold. Psychiatricillnesses can predispose to frostbite through increased risk behavior (14,22). Various medications that affect the circulation,metabolism, and fluid balance may predispose to frostbite (22). Impaired peripheral cold-induced vasodilation (CIVD) and rewarming responses may predict frostbite risk, but the findingsare inconclusive (23,24,61–63).

## Prevention

The primary strategy to reduce frostbite risk is to assess risk,and to respond to it with appropriate mitigation strategies. Thewind chill temperature (WCT) index, which integrates temperature and wind speed (64) provides an estimation of face cooling and cheek frostbite risk (Fig. 1). Exposed fingers willfreeze at a warmer WCT than the cheek (65). Wind markedlyincreases convective heat loss, decreases clothing insulation capacity, and increases evaporative heat loss (66). Frostbite riskcan be based on the WCT index and the period in which ex- posed skin will freeze in more susceptible persons in the population. The risk of frostbite on bare skin is less than 5% when the ambient temperature is above −15°C (5°F), but increasedsafety surveillance is warranted when the WCT index falls be-low −27°C (−17°F), when frostbite can occur in 30 min or less(4). Wet skin cools more rapidly and may increase risk (67). Exposure to volatile liquids (which evaporate easily — such aslight liquid fuels) is of even greater risk. Exercise of sufficient in-tensity increases skin perfusion and reduces skin cooling andcold injury risk (25,67).

Touching or gripping cold material elicits contact cooling and can cause a frostbite injury within a few seconds (26). The degree of skin cooling depends on the surface temperature, type of material, contact duration, and several individualfactors. Human tissue in saltwater freezes at −0.55°C (31°F), whereas seawater freezes at −1.9°C (28.6°F), so frostbite canoccur in very cold seas. Altitudes above 5000 m (18) increasefrostbite risk with the risk potentiated by wind and possibly byfactors, such as dehydration. Many factors may contribute tothis, with low environmental temperatures perhaps combiningwith hypobaric hypoxia (27). In all circumstances, frostbite risk is mitigated through themaintenance of core body temperature, by reducing risk of con-tact freezing (rapid heat loss through a conductor at a temperature below zero), and through a general approach aimed at reducing heat loss with clothing. Thus, skin exposure should beavoided, windproof external layers used, excellent thermal insulation (trapping warmed air, and limiting conduction from the skin) used. Where appropriate, multiple layers can help (*e.g.*, thinner gloves worn under mitts offer dexterity for shortperiods when the hands are removed from those thicker mitts).In extreme conditions, portable heating devices (whether chemical or powered pads) can offer value. In freezing conditions,liquid should be rapidly removed from exposed skin. This is especially important where the liquid might evaporate fast orconduct easily (*e.g.*, some liquid fuels).

## Treatment

If frostbite is suspected, any further cold exposure should beavoided with the casualty placed in a warm, dry environment.Wet clothing should be removed and the injured region protectedfrom direct mechanical injury (*e.g.*, no weight bearing if lowerlimb affected). If hypothermia is present, this should be treatedfirst, with systemic hydration restored and maintained. Routineantibiotic administration should be avoided, as should spontaneous thawing or rewarming through friction or via heat sources (*e.g.*, flame, vehicle engine) (68). The region shouldonly be thawed if refreezing can be prevented (69). Thawingmay initially use the body heat of the casualty or rescuers (*e.g.*,placing the affected region in the axilla). When available, a 37°Cto 39°C (98.6°F to 102.2°F) waterbath should be used until theskin has softened and is reddening (11). Once dried, loose dressings and bandages can be applied. Swelling can lead to bandagestightening and restricting blood flow. Thawing can be painful; ad-ministration of analgesics (non-steroidal anti-inflammatory drugs,paracetamol, sometimes opioids) may be required. Blister integrity should be preserved, and efforts made to prevent secondary infetion. Expert medical evaluation is required (70).

## Nonfreezing Cold Injury

Nonfreezing cold injury (previously referred to as trench foot)often results from exposure to cold-wet conditions causing tissue temperatures to fall below 15°C (59°F) for a prolonged period.The periphery is more commonly affected (not only fingers/toesbut also nose/ears). Unlike frostbite, the tissues do not freeze; in-stead, protracted intense vasoconstriction and associated ischemia and/or reperfusion cause neurovascular damage (71).

Chilblains, a mild form of NFCI, occur following 1 to 5 h ofcold-wet exposure (above freezing) and predominantly affectfinger and toe skin (72). They are small, swollen, itchy, erythematous papules, which may be tender or painful (73). A hyperemic response to rewarming is characterized by red, hot, and swollen skin accompanied by an itching or burning sensation that may persist for several hours. Long-lasting effectsare rare (73). More severe NFCI has not only long affected themilitary (28) but also occurs among athletes, such as ice skaters(74), cyclists (75), divers (76), and long-distance Polar rowers
(77) and is a potential risk for hikers and mountaineers who be-come incapacitated (78).

The “dose” of cold (temperature and duration) required to cause NFCI is not known and varies between and, possibly, within individuals. Most reports of NFCI have involved sev-eral days to weeks of cold exposure (28,75,77,79); however,exposures <24 h can cause NFCI (76,80). Short cold expo- sures may result in NFCI if there is inadequate rewarming and, therefore, prolonged low tissue temperature. The affectedarea is pale, cold, and numb during cold exposure. On rewarming, it becomes cyanotic while remaining cold and numb; it may swell in severe cases. In very mild cases, recovery occurs within a few days with no lasting symptoms (81,82). Inothers, a subsequent hyperemic phase lasts between 2 wk and3 months and is characterized by hot, red, and dry skin with some paresthesia and, in severe cases, blistering (50,81). Theextent of tissue damage can only be assessed after this hyper-emic phase. Chronic symptoms may then occur, lasting froma few months to many years, and include (in varying combinations and severity): cold sensitivity, sensory neuropathy, pain,and hyperhidrosis.

Cold sensitivity is characterized by cool skin temperatureseven in a warm environment and slow rewarming (due to reduced skin blood flow) following local cooling (83,84). In combination with hyperhidrosis, which will increase evaporative cooling, cold sensitivity increases the risk of subsequentcold injury (29,85). Responses to peripheral cooling are di- verse (86); a large proportion of the general population are cold sensitive, some perhaps having subclinical forms of NFCI(84,87–89). Athletes, such as windsurfers (90) and cold-waterswimmers, may develop cold sensitivity through cold expo- sure, although altered sensory thermal sensation or endothelial dysfunction is not generally observed (89).

Individuals from African-Caribbean backgrounds are moresusceptible to NFCI (21,82) as are those with previous cold in-jury (29). Women also may be at greater risk due to their greater rate of hand and foot cooling in the cold (91). The evidence for either dehydration (28,71,77,82,92,93) or smoking(28,30,82,94) increasing the risk of NFCI is equivocal.

A

| Wind (mph) \ Temperature (°F) | Calm | 40 | 35 | 30 | 25 | 20 | 15 | 10 | 5 | 0 | -5 | -10 | -15 | -20 | -25 | -30 | -35 | -40 | -45 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 5 | 36 | 31 | 25 | 19 | 13 | 7 | 1 | -5 | -11 | -16 | -22 | -28 | -34 | -40 | -46 | -52 | -57 | -63 |
| | 10 | 34 | 27 | 21 | 15 | 9 | 3 | -4 | -10 | -16 | -22 | -28 | -35 | -41 | -47 | -53 | -59 | -66 | -72 |
| | 15 | 32 | 25 | 19 | 13 | 6 | 0 | -7 | -13 | -19 | -26 | -32 | -39 | -45 | -51 | -58 | -64 | -71 | -77 |
| | 20 | 30 | 24 | 17 | 11 | 4 | -2 | -9 | -15 | -22 | -29 | -35 | -42 | -48 | -55 | -61 | -68 | -74 | -81 |
| | 25 | 29 | 23 | 16 | 9 | 3 | -4 | -11 | -17 | -24 | -31 | -37 | -44 | -51 | -58 | -64 | -71 | -78 | -84 |
| | 30 | 28 | 22 | 15 | 8 | 1 | -5 | -12 | -19 | -26 | -33 | -39 | -46 | -53 | -60 | -67 | -73 | -80 | -87 |
| | 35 | 28 | 21 | 14 | 7 | 0 | -7 | -14 | -21 | -27 | -34 | -41 | -48 | -55 | -62 | -69 | -76 | -82 | -89 |
| | 40 | 27 | 20 | 13 | 6 | -1 | -8 | -15 | -22 | -29 | -36 | -43 | -50 | -57 | -64 | -71 | -78 | -84 | -91 |
| | 45 | 26 | 19 | 12 | 5 | -2 | -9 | -16 | -23 | -30 | -37 | -44 | -51 | -58 | -65 | -72 | -79 | -86 | -93 |
| | 50 | 26 | 19 | 12 | 4 | -3 | -10 | -17 | -24 | -31 | -38 | -45 | -52 | -60 | -67 | -74 | -81 | -88 | -95 |
| | 55 | 25 | 18 | 11 | 4 | -3 | -11 | -18 | -25 | -32 | -39 | -46 | -54 | -61 | -68 | -75 | -82 | -89 | -97 |
| | 60 | 25 | 17 | 10 | 3 | -4 | -11 | -19 | -26 | -33 | -40 | -48 | -55 | -62 | -69 | -76 | -84 | -91 | -98 |

Frostbite Times: 30 minutes | 10 minutes | 5 minutes

B

| Wind Speed (km/h) | 5 | 0 | -5 | -10 | -15 | -20 | -25 | -30 | -35 | -40 | -45 | -50 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 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 | -30 | -37 | -43 | -49 | -56 | -62 | -68 |
| 25 | 1 | -6 | -12 | -19 | -25 | -32 | -38 | -44 | -51 | -57 | -64 | -70 |
| 30 | 0 | -6 | -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 | -69 | -76 |
| 55 | -2 | -8 | -15 | -22 | -29 | -36 | -43 | -50 | -57 | -63 | -70 | -77 |
| 60 | -2 | -9 | -16 | -23 | -30 | -36 | -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 | -58 | -65 | -72 | -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 |

| FROSTBITE GUIDE |
|---|
| Low risk of frostbite for most people |
| Increasing risk of frostbite for most people in 10 to 30 minutes of exposure |
| High risk for most people in 5 to 10 minutes of exposure |
| High risk for most people in 2 to 5 minutes of exposure |
| High risk for most people in 2 minutes of exposure or less |

Figure 1: WCT Index in °F (A) and °C (B). Frostbite time indicated on both charts is the risk of cheek frostbite in the most susceptible 5% of the population. From (64).

## Prevention

NFCI prevention should focus on keeping the body warm by remaining active; feeling generally cold and being static are NFCI risk factors (82). It also is essential that risk be assessed in the manner suggested for frostbite and, likewise,that tissues are protected from heat loss through conduction, convection, radiation, and evaporation (see above). Of particular note is that wet or damp conditions greatly increase risk,because of the enhanced ability of water to conduct heat, andto drive cooling through evaporation. Appropriate clothing (waterproof, windproof, breathable, and able to maintain its insulation even in wet windy conditions) is essential, as is protection of the hands and feet. Waterproof boots and gloves with breathable membranes to prevent sweat accumulation during periods of high activity and subsequent cooling through evaporation and conduction during periods of rest are required (31). In cold-wet conditions, socks and gloves should be changed frequently to ensure the feet and hands staydry. Restrictive footwear reduces blood flow and increases foot cooling rates and should be avoided (31).

Interindividual NFCI susceptibility varies greatly, even whenclothing, environment, and tasking are identical (28,77). There-fore, risk mitigation needs to be on an individual basis, with anyone reporting feeling cold, complaining of cold extremitiesbeing closely monitored or withdrawn from the event. If NFCIis suspected, individuals should be removed from the cold-wetenvironment to prevent further cooling and enable core rewarming if the individual is hypothermic. The affected feet/hands should be rewarmed slowly (11,50).

## Accidental Hypothermia

Accidental hypothermia is defined as a drop in deep body (core) temperature ($T_{deep}$) to <35°C (95°F) (95,96). Primary hypothermia (commonest type of hypothermia in sports settingsamong young and healthy athletes) results from environmental cold exposure, and secondary hypothermia from factors, suchas exhaustion, trauma, insufficient home heating/insulation, dis-ease and intoxication, advanced age, or multimorbidity (97). Thesigns and symptoms of hypothermia are presented in Table 3.

In primary accidental hypothermia, body heat loss exceeds heat production. The rate of cooling depends on factors, including clothing worn, environmental conditions (*e.g.*, water exposure,temperature, wind), and exercise intensity. Cooling during sedentary cold water (10°C to 16°C) immersion in normothermic individuals ranges from ~1.0 to 1.8°C·h$^{-1}$ (1.8 to 3.2°F·h$^{-1}$) (99–101).It has been measured as high as 9°C·h$^{-1}$ (16.2°F·h$^{-1}$) when buriedin avalanche snow (102,103). In air, physical activity attenuates$T_{deep}$ cooling due to significantly increased heat production com-pared with resting (4). Other factors also impact $T_{deep}$ cooling;for example, shivering heat production is substantially impairedby a central mechanism if hypoglycemia occurs (104). In coldair, hypothermia is most likely to occur if an individual be- comes injured or exhausted and is no longer able to exercise.

## Prevention

When combined with exercise-induced heat production, appropriate clothing provides the greatest protection against hypothermia by reducing convective and evaporative heat lossthrough windproofing and waterproofing, and insulation pro-vided by air trapping (105). Clothing requirements vary withchanges in ambient air temperature, rainfall, and exercise intensity (105,106). In the case of water immersion, the depth of immersion (surface area of body exposed, compressive impact onclothing insulation), water temperature, and movement also will determine clothing requirements. Thermal models estimating whole-body cooling and needed clothing insulation, such asthe Insulation Required (107) and Cold-Weather Ensemble Decision Aid (108,109) predict the amount of clothing needed forindividuals to maintain $T_{deep}$ based on ambient temperatures and exercise intensities. At higher exercise workloads, less clothing is needed to protect against a fall in $T_{deep}$.

Typical cold-weather clothing consists of three layers: an in-ner layer, which is in direct contact with the skin and does notreadily absorb moisture, but wicks moisture to the outer layers where it can evaporate; a middle layer, which provides the pri mary insulation; and an outer layer, which is designed to al- low moisture transfer to the air while repelling wind and rain.Sweating can easily exceed the vapor transfer rate of the outershell layer, causing moisture to accumulate on the inside, evenif the outer layer has substantial venting (*e.g.*, zippers in armpits) to allow moisture to escape. The outer layer should typically not be worn during exercise (unless it is raining or very windy) but should be donned during subsequent rest periods. In group settings, individuals should adjust clothing totheir own physiological responses (*e.g.*, sweating) and not wear a standard amount of clothing. A common problem is that people begin exercising while still wearing clothing layersappropriate for resting conditions, and thus are “overdressed”after initiating exercise. If the combination of environmentalconditions, work intensity, and available clothing suggest thatbody heat content cannot be maintained (*e.g.*, low exercise in-tensity in rainy conditions), then supervision or use of the buddy system should be encouraged. Remaining dry, especiallyfor those exercising in remote regions, is extremely importantand carrying extra clothing that is waterproof, and dry clothing to change into, is important.

## Treatment

Vital signs diminish as cooling progresses (Table 4, [95]). Accurate $T_{deep}$ measurement using rectal or esophageal probesis difficult in a field situation. In such a situation, accidental hypothermia should be diagnosed by measuring tympanic temperature with an insulated thermistor-based probe, allowing readings of <32°C (89.6°F) (113). This can later be confirmedwith a rectal temperature using a low reading thermometer, where and when practical.

Without accurate temperature measurements, the diagnosisand classification of hypothermia must rely on medical historyand clinical findings (*e.g.*, trunk feels cold, quality of vital signs; Table 4). A revised hypothermia staging/classificationhas been proposed (96), which correlates the level of consciousness with the risk of hypothermia-induced cardiac arrest(Table 5). Young and healthy casualties may have vital signsstill present at $T_{deep}$ < 24°C (75.2°F) (115). Signs of breathingor cardiac activity (and/or respiratory and pulse rate) should be sought for at least 1 min because respiratory rates may beas low as 3 to 4 min, pulse

rates as low as 10 to 15 bpm,and pulse volume low and breaths shallow (95,96,115).

Treatment algorithms have been published for patients withaccidental hypothermia (Fig. 2) (95,96,116). Out-of-hospital treatment consists of limiting further cooling. Patients shouldbe removed from wind and water. In a warm shelter, removewet and cold clothing. Out-of-hospital rewarming is almost impossible with limited technical equipment; transport to a hospital should take precedence (see below). In many patients, $T_{deep}$ will continue to fall after rescue (*i.e.*, "afterdrop"). The risk of hypothermia-induced cardiac arrest commences once $T_{deep}$ is <30°C (86°F) in young and healthy casualties; in the elderly and multimorbid the risk increases at $T_{deep}$ < 32°C (89.6°F) (96).

For mildly hypothermic patients in a field environment, warm clothing and drinks with sugar should be given with supervision and active rewarming encouraged. In mild hypothermia cases that recover fully and risk factors are mitigated,there is no need to evacuate. For moderate and severe hypothermia and the critically ill, patients need to be handled verygently (as mechanical impact can trigger cardiac arrest), keptinsulated, passively rewarmed slowly (0.75 to 1.0°C·h$^{-1}$)and evacuated.

Out-of-hospital, casualties with stage 1 hypothermia (Table 5)should be transported to a hospital able to deal with concomitant illnesses or injuries (95,96). With colder patients, the risk of imminent cardiac arrest has to be evaluated (*i.e.*, systolic blood pres-sure <90 mm Hg, ventricular arrhythmia, $T_{deep}$ < 30°C (86°F);Fig. 2). If only one risk factor is present, the patient should betransported to a hospital with the possibility to rewarm themwith extracorporeal life support (ECLS). Without any risk fac-tor of imminent cardiac arrest, the patient can be carefully andgently transported to the closest appropriate hospital and rewarmed actively (*e.g.*, forced warm air). If a patient has suffered a hypothermia-induced cardiac arrest (stage 4), cardiopulmonary resuscitation (CPR) should be initiated immediately.Modifications to CPR have been proposed for hypothermia- induced cardiac arrest patients, for example, at a $T_{deep}$ < 30°C(86°F), no epinephrine and a maximum of three shocks shouldbe attempted (95,96). Transport of a hypothermic patient in cardiac arrest should be directly to a hospital with ECLS rewarming capability.

In the hospital, prognostication of outcome after ECLS rewarming should be performed with specific scores (Hypothermia outcome prediction after extra-corporeal life support (HOPE) or International accidental hypothermia extracorporeal life support (ICE)) (117–119), which are more reliable than the traditional potassium triage (120,121).

Avalanche Burial

During avalanche burial, smaller people lose heat more rap-idly than larger individuals because of a higher surface-area ratio and smaller muscle mass (122). Regarding cooling rate,the amount of subcutaneous adipose tissue and insulation provided by clothing also are important. For example, while wearing a thin monolayer garment, commonly used by back-country skiing athletes during ascent, body core cooling can approach 9°C·h$^{-1}$ during an avalanche burial. With thick multilayer clothing ensembles, including helmet and gloves,used in downhill skiing, cooling is slower (123).

Of all avalanche-buried persons, 10% to 20% die in the first 30 min from trauma to the head, cervical and thoracic spine, or from multiple trauma (124). More than 60% die from asphyxia (*i.e.*, lack of oxygen), most commonly withinthe first 35 min of burial because either the airways are obstructed or snow in front of the mouth and nose inhibitsair inhalation. The longer someone is buried following an avalanche, the less chance of survival (124,125). Data in European skiers from 1981 to 1991 (126,127) suggest three phases of avalanche burial (survival, asphyxiation, waiting) before rescue. For survival, 93% of avalanche victims are stillalive in the first 15 to 20 min and with asphyxiation, 65% dieduring the next 15 min due to freezing snow caused by breath-ing leading to a limited oxygen availability. Survival to 45 minand beyond suggests an open-air pocket exists (*i.e.*, patent air-ways with space in front of mouth and nose with access to open air). These casualties will survive until an avalanche- specific combination of hypothermia, hypoxia, and hypercapnia sets in (128). Less than 20% survive for more than 2 h (129). Climate and topography affect the survival from avalanche burial (126). In humid climates (*e.g.*, maritime coast),snow is denser, and asphyxia has an earlier onset than in the Swiss Alps with a continental climate. Skiing in slightly forested or rocky terrain results in more fatalities due to trauma.

Reduced burial depth is positively correlated with survival.Only 4.4% of partially buried casualties (*i.e.*, head and chestoutside of the snow) compared with 51.3% of fully buried casualties (*i.e.*, head and chest below the snow) die (130).An avalanche airbag reduces the risk of full burial and also may reduce burial depth. The reduction in mortality is less when the airbag does not inflate: in one study, noninflation occurred in 20% of use, 60% of which was attributed to a deployment failure by the user (131). With risk of avalanches, winter sport athletes who are outside protected slopes shouldbe equipped with, and trained in the use of, an avalanche airbag, an avalanche transceiver, a probe, and a metal-headedshovel. Companions should quickly track the location with an avalanche transceiver, probe the exact location and depth,and excavate the buried casualty. Using digital instead of ana-log avalanche transceivers, attending avalanche rescue courseson correct tracking and probing, as well as regular training indeploying an avalanche airbag and working in coordinated groups will allow faster extrication. In mountain regions closeto urban centers, helicopter rescue has revolutionized avalancherescue because of fast transport times. Still, professional rescue should not be expected on scene within the first 20 min. Thus,rescue by peers on-site is of utmost importance to increase survival chances.

Once extricated, patients are treated according to specific avalanche resuscitation guidelines (96,118,119). In normo-thermic patients, asphyxia triggered cardiac arrest can only be survived for a few minutes and has a poor outcome. In hypothermia triggered cardiac arrest (usually <30°C; 86°F), out-come is substantially better (96,132,133).

Snow Blindness

The structures of the eye are vulnerable to damage from exposure to ultraviolet (UV) light, with risk increasing at high altitudes

(rising 4% per 300 m ascent). Being protected by the brow, nose, and upper lid, the eye is mainly exposed to UV- light that is reflected (emphasizing a need for lateral eye protection). The reflective incidence of water and snow are two-fold and eightfold greater than grass. In a snowy environmentat 2000 m, UV exposure is doubled (134,135).

"Snow blindness" results from acute UV ocular injury. Thedegree of conjunctival and corneal injury ("ultraviolet keratitis") depends on energy intensity and exposure duration. Limbic in-jury causes pain when trying to focus the lens. Symptoms begin4 to 10h after exposure and range from a "gritty" sensation tosevere pain, blurred vision, uncontrolled blinking, eye-watering, photophobia, and blepharospasm. Symptoms last <48 h, or several days if severe. Injury can be compounded by corneal swelling, which results from altitude-and wind-related hypoxia and corneal evaporative drying, with hypoxia worsenedby contact lens use (135,136). A fluorescein eye stain test canlocate corneal injuries.

Prevention

Sunglasses or goggles with side protection, which absorb >95% of all UV light, should be worn. Soft contact lenses, which block UV-light and cover the pupil and limbus, offer good protection. Brimmed hats offer additional shade.

Treatment

Close eyes and rest until pain eases. In a first step, cool gentlythrough the closed eyelid (135). In a next step use oral analgesics(*e.g.*, paracetamol 1g, four time a day). If available, administer atopical lubricant (*e.g.*, antioxidant artificial tears), and apply nonsteroidal anti-inflammatory eyedrops (*e.g.*, diclofenac 0.1%). Topical anesthetics slow corneal recovery and shouldnot be used outside emergency situations (*e.g.*, descent fromhigh altitude). Consider topical antibiotics in severe cases to prevent infection. Cycloplegic eye-drops (cyclopentolate 1%)may relieve pain but impair vision (135,136).

Cold Water

The great cooling power of water means that some of the re-sponses described for cold air (*e.g.*, hypothermia) also occur incold water, but sooner. Humans cool four to five times fasterin cold water than in air at the same temperature (137).

There are four stages of immersion in cold water, each asso-ciated with specific hazards and each related to the cooling of different body tissues. Rapid skin cooling on initial immersionstimulates the cutaneous cold receptors; their dynamic re- sponse elicits the "cold shock," a response not evoked by theslower rates of skin cooling in air. Cold shock is regarded asthe most dangerous response caused by cold water immersionand affects men and women to a similar extent (138,139). Thegreatest responses are observed at water temperatures between10°C and 15°C (50°F to 59°F) for lightly clothed (swim-suited)individuals and include a "gasp" response, hyperventilation, hypertension, and increased cardiac workload (140). These can be precursors to drowning and cardiovascular issues. Theinitial gasp response is 2 to 3 L (141,142), larger than the lethalvolume of seawater for drowning (1.5 L, [143]). The cold shockresponse peaks in the first 30 s of immersion and usually abatesover the next 90 s as the peripheral cold receptors adapt to thenew skin temperature. There is a significant possibility of aspi-rating water during initial immersion when conscious controlof breathing has been lost. In situations where the face also isimmersed on initial immersion, coactivation of sympathetic and parasympathetic inputs to the heart can produce "auto- nomic conflict," resulting in potentially fatal arrhythmias in avariety of sporting situations (*e.g.*, open water swimming, tri-athlon [144]).

After skin, the next body tissues to be affected by cooling arethe superficial nerves and muscle. The arms are particularly sus-ceptible to cooling due to their anthropometry (thin cylinders),anatomy (superficial nerves and muscles), and physiology (reli-ance on blood flow for warming) (122). Within about 20 minof immersion in water of 12°C (54°F) peripheral neuromuscularcooling can significantly impair manual dexterity, grip strengthand coordination, impacting swimming ability (122,145).

Approximately 60% of cold-water immersion deaths occurwithin the first minutes of immersion, long before hypother-mia occurs (144). Under normal circumstances, adult humanswill not become hypothermic in under 30 min in even the coldest water.

Prevention/Protection/Treatment

The cold shock response demonstrates temporal summa- tion, with a greater response being observed with faster ratesof entry into cold water (146). Those entering cold water areadvised to rest with their airway clear of the water until they have regained control of their breathing ("float first"). Pro-longed head-in breath holding should be avoided on initial im-mersion. For open cold water swimming events, swimmersshould adapt to the water just before commencing the swim(or have in-water starts); this reduces the chance of aspiratingwater and makes the coordination of the swim stroke and breathing easier.

Protection can be physiological or technological. Physiological protection against cold shock can be achieved by cold ha-bituation, with as few as six 2-min head-out cold water im mersions over 3 d reducing the cold shock response by 50% by the last immersion (147). A reduction of 25% is still apparent 14 months later (148). Although habituation will reduce the ventilatory response on immersion, this may not translateto an improved swimming capacity (149).

Importantly, repeated immersion in cold water does not protect against neuromuscular incapacitation from peripheral cooling; protection for this response can only be achieved bylimiting exposure or technological protection with protective clothing. In terms of swim failure, it appears that the upper arms (triceps region) are the most important region to protect (145,150). However, insulation of the upper arms during a simulated survival swim in 10°C water maintained warmer arm skin temperature and swimming technique but did not improve swimming duration or distance (151). Most sports persons use wet suits rather than dry suits. The primary determinants of the protection provided by a wet suit are its fit andthickness. The fit

should be as tight as possible commensuratewith sporting performance, and the thickness varies betweenthe torso and the arms, depending on the sport and requirements for regional flexibility. The minimum recommended water temperature for triathlon racing is 12°C (54°F) inwetsuits and 16°C (61°F) without wetsuits (152,153). It is recommended that water temperatures below 18°C (64°F) are too cold for elite marathon swim racing without wet suit protection (154). Fédération Internationale de Natation rules were changed in 2017 to make wetsuits compulsory below 18°C (64°F) and optional below 20°C (68°F) (155).

Protection also can be afforded by appropriate event organization. Swimming events, including mass starts, can be orga-nized and supervised to minimize the chances of cardiac andrespiratory-related problems (156). Those providing safety cover should be aware of the increased likelihood of cold- related problems in the first (due to cold shock response) and last (due to fatigue) minutes of an event and should be trained to recognize impending swim failure (145).

Basic life support compressions and ventilations (2 rescuebreaths then CPR at 30:2 ratio) should be used and for drown-ing victims, high concentrations of inspired oxygen given as soon as possible. All those suspected of aspirating water shouldbe evacuated to hospital. For hypothermic casualties, follow theadvice given in the section on “Accidental Hypothermia.”

Table 3.

Signs and symptoms of hypothermia at different levels (98).

| Mild (32–35°C; 89.6–95°F) | Moderate (28–31°C; 82.4–88°F) | Severe (<28°C; <82.4°F) |
|---|---|---|
| Cold extremities | Apathy, poor judgment | Inappropriate behavior |
| Shivering | Reduced shivering | Total loss of shivering |
| Tachycardia | Weakness and drowsiness | Cardiac arrhythmias |
| Tachypnea | Slurred speech and amnesia | Pulmonary edema |
| Urinary urgency | Dehydration | Hypotension and bradycardia |
| Mild incoordination | Decreased coordination or clumsiness | Reduced level of consciousness |
| | Fatigue | Muscle rigidity |

Table 4.

The two main clinical classification systems for accidental hypothermia: The original Swiss system (110) and the wilderness medical society classification (111).

| Swiss System (110) | | | WMS (111) | | |
|---|---|---|---|---|---|
| Category | Clinical Findings | Estimated Core Temperature (°C, °F) | Category | Clinical Findings | Estimated Core Temperature (°C; °F) |
| Stage 1 | Clear consciousness with shivering | 35°C to 32°C | Mild | Normal mental status, shivering, but not functioning normally/ able to care for self | 35°C to 32°C |
| | | 95°F to 89.6°F | | | 95°F to 89.6°F |
| Stage 2 | Impaired consciousness without shivering | <32°C to 28°C | Moderate | Abnormal mental status with shivering,or abnormal mental status without shivering, but conscious | 32°C to 28°C |
| | | <89.6°F to 82.4°F | | | <89.6°F to 82.4°F |
| Stage 3 | Unconsciousness | <28°C to 24°C | Severe/profound | Unconscious | <28°C |
| | | <82.4°F to 75.2°F | | | <82.4°F |
| Stage 4 | Apparent death | 24°C to 11.8°C | | | |
| | | 75.2°F to 53.2°F | | | |
| Stage 5 | Death due to irreversible hypothermia | <11.8°C | | | |
| | | <53.2°F | | | |

Core temperature data from stages 4 and 5 from Mroczek et al. (112).WMS, Wilderness Medical Society.

Table 5.

Revised Swiss system for staging of accidental hypothermia (114).

| | Stage 1 | Stage 2 | Stage 3 | Stage 4 |
|---|---|---|---|---|
| **Clinical findings**[a] | "Alert" from AVPU | "Verbal" from AVPU | "Painful" or "Unconscious" from AVPU<br>Vital signs present | "Unconscious" from AVPU<br>**AND**<br>No detectable vital signs[b] |
| **Risk of cardiac arrest**[c] | **Low** | **Moderate** | **High** | **Hypothermic cardiac arrest** |

AVPU denotes Alert, Verbal, Painful and Unconscious, respectively.

[a] In the Revised Swiss System, "Alert" corresponds to a GCS score of 15; "Verbal" corresponds to a GCS score of 9 to 14, including confused patients; "Painful" and "Unconscious" correspond to a GCS score <9. While shivering is not used as a stage-defining sign in the Revised Swiss System, its presence usually means that the temperature is >30°C (86°F), a temperature at which hypothermic CA is unlikely to occur.

[b] No respiration, no palpable carotid or femoral pulse, no measurable blood pressure. Check for signs of life (pulse and, especially, respiration) for up to 1 min.

[c] The transition of colors between stages represents the overlap of patients within groups. The estimated risk of cardiac arrest is based on accidental hypothermia being the only cause of the clinical findings. If other conditions impair consciousness, such as asphyxia, intoxication, high altitude cerebral edema or trauma, the revised Swiss System may falsely predict a higher risk of cardiac arrest due to hypothermia. Caution should be taken if a patient remains "alert" or "verbal" showing signs of hemodynamic or respiratory instability like bradycardia, bradypnea, hypotension because this may suggest transition to a stage with higher risk of cardiac arrest.

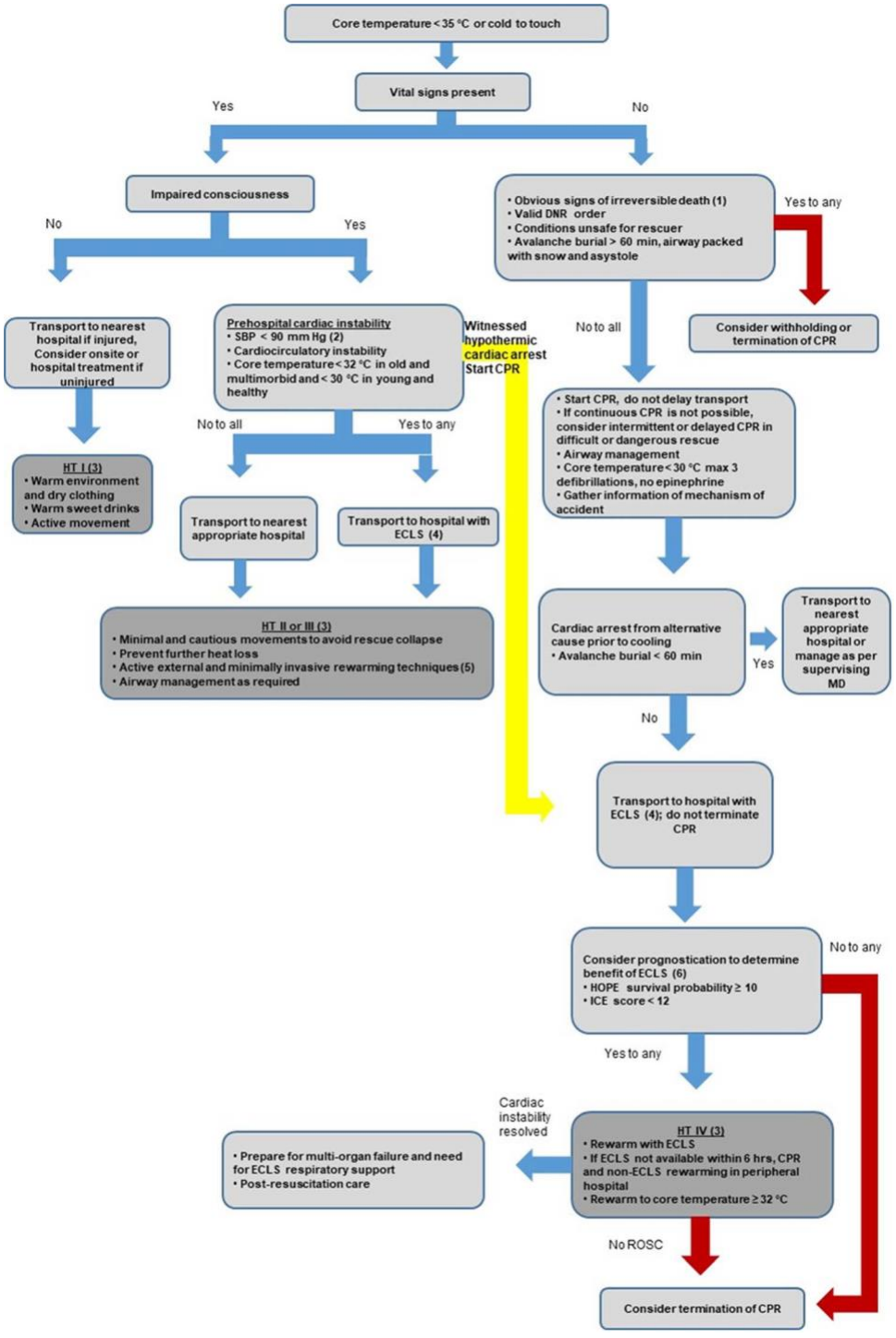


**Figure 2:** Treatment algorithm for patients with accidental hypothermia, from (96). Definitions of parenthetical numbers are: (1) decapitation, truncal transection, whole body decomposed, or whole body frozen solid (chest wall not compressible); (2) SBP < 90 mm Hg is a reasonable prehospital estimate of cardiocirculatory instability but for in-hospital decisions, the minimum sufficient circulation for a deeply hypothermic pa-tient (*e.g.,* <28°C) has not been defined; (3) Swiss staging of accidental hypothermia; (4) direct transport to an ECMO center is recommendedin an arrested hypothermic patient. In remote areas, transport decisions should balance the risk of increased transport time with the potential ben- efit of treatment in an ECLS center (*e.g.*, 6 h). (5) Warm environment, chemical, electrical, or forced air heating packs or blankets, and warm IV fluids (38°C to 42°C). In case of cardiac instability refractory to medical management, consider rewarming with ECLS.(6) If the decision is madeto stop at an intermediate hospital to measure serum potassium, a hospital en route to an ECLS center should be chosen. HOPE and ICE scoresshould not be used in children; instead consider expert consultation. DNR, do not resuscitate HT, hypothermia, MD, medical doctor, ROSC, return of spontaneous circulation, SBP, systolic blood pressure.

Cold and Performance

Cold air and water exposure can potentially have deleterious effects on aerobic and strength performance (157–163). Interested readers are referred to previous reviews of the im- pact of cold exposure on exercise performance and the physiological mechanisms responsible for cold-related changes inperformance (122,164,165)

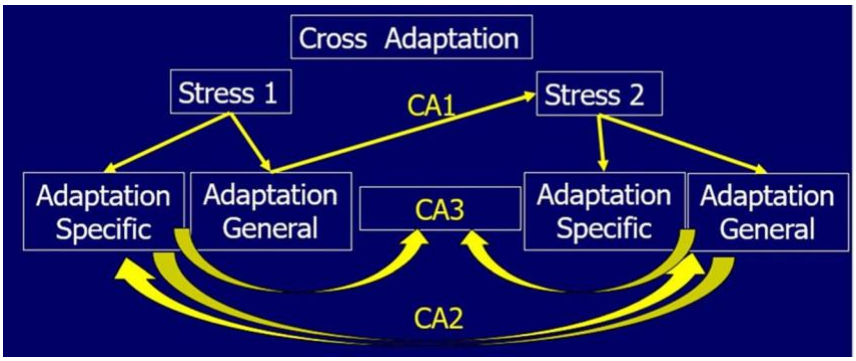


Figure 3: Theoretical overview of cross-adaptation (CA). CA1, adaptation to one stimulus provides cross-tolerance to another. CA2, adaptation to one stressor enhances adaptation to another stressor. CA3, combined adaptive effects of two stimuli providing beneficial responses to a third variable. The cross-adaptive effect may *"*general*"* via some common pathway involving for example the autonomic nervous system or pathwaysinvolved in cellular tolerance, or *"*specific*"* involving a more specific response to a stimulus such as shivering or vasoconstriction.

Decreased muscle temperatures ($T_{muscle}$) lower $\dot{V}O_{2max}$, ex-ercise time, and power/sprint ability. For every 1°C fall in muscle temperature, there is a 4% to 6% decline in these markers of performance (166–168). For example, an 8°C de-crease in $T_{muscle}$ decreases power output by 31% and maximalvoluntary contraction by 19% (169). Low $T_{muscle}$ (28°C to 29°C) also cause higher muscle lactate levels in both type 1 and type 2 muscle fibers (170,171), and overall, blood lactatelevels increase during exercise to a greater extent in cold com-pared with temperate environments (157,172–177) suggest- ing a greater reliance on anaerobic metabolism. Maximal heart rate is lower by 10 to 30 bpm when $T_{deep}$ is loweredby 0.5°C to 2.0°C (178) and cold water decreases leg muscleblood flow during exercise (173,179).

In cold air, where there is little change in $T_{deep}$ or $T_{muscle}$, there is a lack of consensus on whether aerobic performancedeclines: studies have demonstrated impairment (158–161), improvement (180,181), or no change (182,183). Two studies have examined, systematically, whether air temperature af-

fects performance. Cycling time to exhaustion at 70% $\dot{V}O_{2max}$ was longest at an ambient temperature ($T_{amb}$) of11°C while wearing shorts/t-shirt (158) with decrements seen

at 4°C and 31°C. While wearing cross-country ski uniforms,Sandsund et al. (160) observed maximal running performanceat $T_{amb}$ between −4°C (24.8°F) and 1°C (33.8°F), with perfor-mance reduced at −14°C (6.8°F) and warmer $T_{amb}$. The lower$T_{amb}$ in the running study can perhaps be attributed to the skiuniform conferring greater thermal protection and the higher exercise intensity. However, it should be noted that if more clothing is needed to protect against environmental cold expo-sure, this could reduce performance due to increased energy de-mands caused by heavy clothing, resistance to movement, andother equipment (4,184). Upper-body performance in cross- country skiers also is reduced at colder $T_{amb}$ (185,186). Coldtemperatures also can impact biomechanics and gait, increasingthe energy demands of exercise (187). Furthermore, athletes need to be cognizant of terrain factors (*i.e.*, ice, snow cover) thatcan cause slipping and coordination issues. Proper nutrition and hydration are important for maintaining performance in cold-weather environments. Practical recommendations in- clude maintaining adequate carbohydrate stores and monitor-ing fluid intake/output by tracking body mass changes and urine output/color (188).

### Combined Stressors

In the natural world, it is rare for stressors, such as heat andhumidity, cold and altitude, and cold and hyperbaric stress,not to be combined or experienced sequentially. However,largely because of the way subject areas are organized, the im-pact of combined stressors on human responses has receivedmuch less attention than the impact of isolated stressors. Be-tween 1948 and 2012, there were only 14 studies examining these areas with human participants (189). Since 2012, the num-ber of studies has increased dramatically and the importance of this area of research for human performance, health, anddisease is beginning to be realized (190). The areas of com-bined stressors not only include the beneficial or detrimental ef-fects of combined stressors, such as cold and hypoxia, but alsoinclude cross-adaptation and cross-tolerance between such stressors (Fig. 3), at both the systemic and cellular levels[1].

Focusing on the combination of cold and altitude (hypoxia)(191), cold-induced thermogenesis is reduced in hypoxia (192),and $T_{deep}$ falls faster (193). During cooling and warming, thevasoconstrictor response can occur earlier and be released laterif hypoxia is combined with cold, thereby increasing the "dose"of cold experienced and the likelihood of cold injuries (193,194), although this remains a matter for debate (195).

Adaptation to the initial responses to cold water immersionreduces the subsequent responses to a hypoxic exposure (196) including plasma epinephrine and norepinephrine, sympa- thetic nervous system response, heart rate, ventilation, oxygen consumption, and carbon dioxide production, as well as symptoms of hypoxia and their severity (196).

In contrast to the clear interaction between cold and hyp- oxia, it has been reported that cold acclimation does not alterthe physiological or perceptual responses during subsequent exercise in the heat (197). The area of combined stressors is clearly fertile ground for further investigation with regard tothe avoidance of injuries in the cold.

## Conclusions

Coaches, athletes, medical personnel, and officials need to understand the physiology, pathophysiology, prevention, protection, and treatment of cold-related impacts on perfor- mance. From this understanding come optimal interventionsfor the maintenance of performance and avoidance of frost- bite, nonfreezing cold injuries, hypothermia, drowning, and other medical events.

Click here (SDC link needed to PowerPoint file, http://links.lww.com/CSMR/A122) to download a slide deck that summarizes this ACSM Expert Consensus Statement on Injury Prevention and Exercise Performance during Cold-Weather Exercise.

[1]Cross-adaptation: "Adaptations made in response to one environmentalstressor are beneficial in another" Cross-acclimatization: "Adaptation de-rived from a natural/terrestrial environment" Cross-acclimation: "Adaptation derived from a simulated environment" Cross-tolerance: single or re-peated exposures to a stressor eliciting a positive adaptive response in cellular and molecular pathways during a subsequent exposure to a differentstressor (Gibson, 2017).

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