ACSM Expert Consensus Statement: Injury Prevention and Exercise Performance during Cold-Weather Exercise

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Summary

Cold injury can result from exercising at low temperatures, and can impair exercise performance, or cause lifelong debility or death. This Consensus Statement 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 make the text more focused and accessible.

This statement addresses the deleterious aspects of exposure 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 papers on the detailed physiological responses to acute and chronic cold exposure and effects of cold acclimatization (2, 3). This statement updates and replaces the ACSM position statement published in 2006, entitled “Prevention of Cold Injuries during Exercise” (4). As an official pronouncement of the College, it reflects the College’s position on the scientific and clinical aspects of 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 cold is a leading causes of death among people engaged 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, non-freezing cold injury, hypothermia, avalanche burial, snow blindness, drowning and sudden cardiac death. In addition, cold can constitute part of a combined environmental threat, for example in combination with hypoxia in high mountains. It follows that an understanding of the impact of cold environments and approaches to mitigate these threats is essential for those hoping to perform in cold conditions.

Cold Air (Frostbite)

Frostbite is a direct freezing injury occurring when the skin surface freezes in saltwater at ~ -0.55 °C (31 °F) (6) and in air below -3 °C (26.6 °F) (7-9). Exposed tissues with poor perfusion are most commonly affected (hands, feet, head) (10-12). Exposure times for injury vary from seconds to hours depending on the type and intensity of cold exposure, degree of physical activity, protective clothing and various individual factors (Table 1) (13). At sub-freezing tissue temperatures, extracellular ice crystals form in susceptible tissue, leading to cellular mechanical damage and increased osmotic pressure, causing inflammation, microvascular thrombosis, ischemia and hypoxia. Formation of intracellular ice crystals may then occur. Thawing increases tissue edema and provokes an inflammatory response and reperfusion injury (11). Frostbite is classified as described in Table 2.

Skin numbness is a sign of a heightened cold injury risk. A pale spot on the skin indicates superficial cold injury, which is characterized by partial skin freezing and mild edema (14). 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 rewarmed unless refreezing can be avoided (15).

Frostbite occurrence ranges from 7-11 % among the general population in Scandinavian countries (10, 16-18). It also occurs in military training (19-21). It is more common in rural, northern climates (16, 22) , in occupations involving high physical strain and extended cold exposure (18); and in leisure/ sporting activities such as mountaineering (23-26), cold climate hiking (27), use of all-terrain vehicles in the cold or of snowmobiles (22); and sports activities generating high wind speed, such as alpine skiing or sledding (28), or associated with prolonged stationary posture, such as kite skiing and hang gliding (29, 30). A recent study concluded that the incidence of frostbite injuries in the Austrian Alps is low (24) , mainly due to better awareness and clothing.

Age (31), sex (10, 14, 16, 18, 22, 24), and ethnic background (32, 33) affect frostbite risk (Table 1). Diseases affecting neural, vascular and metabolic functions and related tissue perfusion and microvascular function, as well as metabolic heat production may also increase frostbite risk. Autonomic and peripheral neuropathies (e.g. diabetes) impair neural control and thermal sensations (34), 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. Endocrine conditions (e.g. hypothyroidism, hypopituitarism, adrenal insufficiency) can decrease metabolic heat production in the cold. Psychiatric illnesses can predispose to frostbite through increased risk behavior (18, 34). Various medications that affect the circulation, metabolism and fluid balance may predispose to frostbite (34). Impaired peripheral cold-induced

vasodilation (CIVD) and rewarming responses may predict frostbite risk, but the findings are inconclusive (35-39).

Prevention

The primary strategy to reduce frostbite risk is to assess risk, and to respond to it with appropriate mitigation strategies. The wind chill temperature (WCT) index, which integrates temperature and wind speed (40) and provides an estimation of face cooling and cheek frostbite risk (Figure 1). Exposed fingers will freeze at a warmer WCT than the cheek (41). Wind markedly increases convective heat loss, decreases clothing insulation capacity, and increases evaporative heat loss (42). Frostbite risk can be based upon the WCT index and the period of time in which exposed 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 increased safety surveillance is warranted when the WCT index falls below -27 °C (-17 °F), when frostbite can occur in 30 min or less (4). Wet skin cools more rapidly and may increase risk (43). Exposure to volatile liquids (which evaporate easily - such as light liquid fuels) is of even greater risk. Exercise of sufficient intensity increases skin perfusion and reduces skin cooling and cold injury risk (43, 44).

Touching or gripping cold material elicits contact cooling and can cause a frostbite injury within a few seconds (45). The degree of skin cooling depends on the surface temperature, type of material, contact duration and several individual factors. Human tissue in saltwater freezes at -0.55 °C (31 °F), whereas seawater freezes at -1.9 °C (28.6 °F), so frostbite can occur in very cold seas. Altitudes above 5000 m (26) increase frostbite risk with the risk potentiated by wind and perhaps by factors

such as dehydration. Many factors may contribute to this, with low environmental temperatures perhaps combining with hypobaric hypoxia. (46).

In all circumstances, frostbite risk is mitigated through the maintenance of core body temperature; by reducing risk of contact 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 be avoided, windproof external layers utilized, 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 short periods 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 or conduct easily (e.g. some liquid fuels).

Treatment

If frostbite is suspected, any further cold exposure should be avoided with the casualty placed in a warm, dry environment. Wet clothing should be removed and the injured region protected from direct mechanical injury (e.g. no weight bearing if lower limb affected). If hypothermia is present, this should be treated first, with systemic hydration restored and maintained. Routine antibiotic administration should be avoided, as should spontaneous thawing or rewarming through friction or via heat sources (e.g., flame, vehicle engine) (47). The region should only be thawed if refreezing can be prevented (48). Thawing may initially use the body heat of the casualty or rescuers (e.g. placing the affected region in the axilla). When available, a 37-39 °C (98.6-102.2 °F) water-bath should be used until the skin has softened and is reddening (11). Once dried, loose dressings and bandages can be applied.

Swelling can lead to bandages tightening and restricting blood flow. Thawing can be painful; administration of analgesics (NSAIDs, paracetamol, sometimes opioids) may be required. Blister integrity should be preserved, and efforts made to prevent secondary infection. Expert medical evaluation is required (49).

Non-freezing cold injury

Non-freezing cold injury (NFCI, previously referred to as trenchfoot) 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 (fingers/toes, but also nose/ears). Unlike frostbite, the tissues do not freeze; instead, protracted intense vasoconstriction and associated ischemia and/or reperfusion cause neurovascular damage (50).

Chilblains, a mild form of NFCI, occur following 1-5 hours of cold-wet exposure (above freezing) and predominantly affect finger and toe skin (51). They are small, swollen, itchy, erythematous papules which may be tender or painful (52). 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 effects are rare (52). More severe NFCI has long affected the military (53), but also occurs among athletes, such as ice skaters (54), cyclists (55), divers (56) and long distance Polar rowers (57) and is a potential risk for hikers and mountaineers who become incapacitated (58).

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 several days to weeks of cold exposure (53, 55, 57, 59); however exposures <24 hours can cause NFCI (56, 60). Short cold exposures may result in

NFCI if there is inadequate rewarming and therefore prolonged low tissue temperature. The affected area 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 (61, 62). In others, a subsequent hyperemic phase lasts between 2 weeks and 3 months, and is characterized by hot, red and dry skin with some paresthesia and, in severe cases, blistering (14, 61). The extent of tissue damage can only be assessed after this hyperemic phase. Chronic symptoms may then occur, lasting from a 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 temperatures even in a warm environment and slow rewarming (due to reduced skin blood flow) following local cooling (63, 64). In combination with hyperhidrosis, which will increase evaporative cooling, cold sensitivity increases the risk of subsequent cold injury (65, 66). Responses to peripheral cooling are diverse (67); a large proportion of the general population are cold sensitive, some perhaps having subclinical forms of NFCI (64, 68-70). Athletes, such as windsurfers (71) and cold-water swimmers may develop cold sensitivity through cold exposure, although altered sensory thermal sensation or endothelial dysfunction is not generally observed (70).

Individuals from African-Caribbean backgrounds are more susceptible to NFCI (33, 62) as are those with previous cold injury (66). Women may also be at greater risk due to their greater rate of hand and foot cooling in the cold (72). The evidence for either dehydration (50, 53, 57, 62, 73, 74) or smoking (53, 62, 75, 76) increasing the risk of NFCI is equivocal.

Prevention

NFCI prevention should focus on keeping the body warm by remaining active; feeling generally cold and being static are NFCI risk factors (62). It is also 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, due to the enhanced ability of water to conduct heat, and to 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 (77). In cold-wet conditions, socks and gloves should be changed frequently to ensure the feet and hands stay dry. Restrictive footwear reduces blood flow and increases foot cooling rates and should be avoided (77).

Inter-individual NFCI susceptibility varies greatly, even when clothing, environment and tasking are identical (53, 57). Therefore, risk mitigation needs to be on an individual basis, with anyone reporting feeling cold, complaining of cold extremities being closely monitored or withdrawn from the event. If NFCI is suspected, individuals should be removed from the cold-wet environment to prevent further cooling and enable core rewarming if the individual is considered to be hypothermic. The affected feet/hands should be rewarmed slowly (11, 14).

Accidental Hypothermia

Accidental hypothermia is defined as a drop in deep body (core) temperature (T_{deep}) to <35 °C (95 °F) (78, 79). Primary Hypothermia (commonest type of

hypothermia in sports settings among young and healthy athletes) results from environmental cold exposure, and Secondary hypothermia from factors such as exhaustion, trauma, insufficient home heating/insulation, disease and intoxication, advanced age or multimorbidity (80).The signs 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 (10-16 °C) immersion in normothermic individuals ranges from ~1.0 to 1.8 °C·h^{-1} (1.8 - 3.2 °F·h^{-1}) (81-83). It has been measured as high as 9 °C·h^{-1} (16.2 °F·h^{-1}) when buried in avalanche snow (84, 85). In air, physical activity attenuates T_{deep} cooling due to significantly increased heat production compared to resting (4). Other factors also impact T_{deep} cooling; for example, shivering heat production is substantially impaired by a central mechanism if hypoglycemia occurs (86). In cold air, hypothermia is most likely to occur if an individual becomes 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 loss through wind- and waterproofing, and insulation provided by air trapping (87). Clothing requirements vary with changes in ambient air temperature, rainfall and exercise intensity (87, 88). In the case of water immersion, the depth of immersion, water temperature and movement will also determine clothing requirements. Thermal models estimating whole-body cooling and needed

clothing insulation such as the Insulation Required (IREQ) (89) and Cold-Weather Ensemble Decision Aid (90, 91) predict the amount of clothing needed for individuals 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 inner layer, which is in direct contact with the skin and does not readily absorb moisture, but wicks moisture to the outer layers where it can evaporate, a middle layer, which provides the primary insulation and an outer layer, which is designed to allow moisture transfer to the air, while repelling wind and rain. Sweating can easily exceed the vapor transfer rate of the outer shell layer, causing moisture to accumulate on the inside, even if 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 to their 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 layers appropriate for resting conditions, and thus are “overdressed” after initiating exercise. If the combination of environmental conditions, work intensity, and available clothing suggest that body heat content cannot be maintained (e.g., low exercise intensity in rainy conditions), then supervision or use of the buddy system should be encouraged. Remaining dry, especially for those exercising in remote regions, is extremely important and carrying extra clothing that is waterproof, and dry clothing to change into, is important.

Treatment

Vital signs diminish as cooling progresses (Table 4, (78)). Accurate T_{deep} measurement using rectal or esophageal probes is 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) (92). This can later be confirmed with a rectal temperature using a low reading thermometer, where and when practical.

Without accurate temperature measurements, the diagnosis and classification of hypothermia must rely on medical history and clinical findings (e.g. trunk feels cold, quality of vital signs; Table 4). A revised hypothermia staging/classification has been proposed (79), which correlates the level of consciousness with the risk of hypothermia-induced cardiac arrest (Table 5). Young and healthy casualties may have vital signs still present at T_{deep} <24 °C (75.2 °F) (93). Signs of breathing or cardiac activity (and/or respiratory and pulse rate) should be sought for at least 1 minute because respiratory rates may be as low as 3-4·min^{-1}, pulse rates as low as 10-15 beats·$min^{-1,}$ and pulse volume low and breaths shallow (78, 79, 93).

Treatment algorithms have been published for patients with accidental hypothermia (Figure 2) (78, 79, 94). Out-of-hospital treatment consists of limiting further cooling. Patients should be removed from wind and water. In a warm shelter, remove wet and cold clothing. Out-of-hospital rewarming is almost impossible with limited technical equipment; transport to a hospital this 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) (79).

For mildly hypothermic patients in a field environment, warm clothing and drinks with sugar should be given with supervision and active re-warming encouraged. For moderate and severe hypothermia and the critically ill, patients need to be handled very gently (as mechanical impact can trigger cardiac arrest), kept insulated, passively rewarmed slowly (0.75-1.0 ˚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 (78, 79). With colder patients, the risk of imminent cardiac arrest has to be evaluated (i.e. systolic blood pressure <90 mmHg, ventricular arrhythmia, T_{deep} <30 °C (86 °F); Figure 2). If only one risk factor is present the patient should be transported to a hospital with the possibility to rewarm them with extracorporeal life support (ECLS). Without any risk factor of imminent cardiac arrest, the patient can be carefully and gently 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, e.g. at a T_{deep} <30 °C (86 °F), no epinephrine and a maximum of three shocks should be attempted (78, 79). 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 (HOPE or ICE) (95-97), which are more reliable than the traditional potassium triage (98, 99).

Avalanche Burial

During avalanche burial, smaller people lose heat more rapidly than larger individuals due to a higher surface-area ratio and smaller muscle mass (100). With regard to cooling rate, the amount of subcutaneous adipose tissue and insulation provided by clothing are also important. For example, while wearing a thin mono-layer garment, commonly used by backcountry skiing athletes during ascent, body core cooling can approach 9 $°C \cdot h^{-1}$ during an avalanche burial. With thick multi-layer clothing ensembles, including helmet and gloves, used in downhill skiing, cooling is slower (101).

Of all avalanche-buried persons, 10-20 % die in the first 30 minutes from trauma to the head, cervical and thoracic spine, or from multiple trauma (102). More than 60 % die from asphyxia (i.e. lack of oxygen), most commonly within the first 35 minutes of burial because either the airways are obstructed, or snow in front of the mouth and nose inhibits air inhalation. The longer someone is buried following an avalanche, the less chance of survival (102, 103). Data in European skiers from 1981-1991 (104, 105) suggest 3 phases of avalanche burial (survival, asphyxiation, waiting) before rescue. For survival, 93 % of avalanche victims are still alive in the first 15-20 minutes and with asphyxiation, 65 % die during the next 15 minutes due to freezing snow caused by breathing leading to a limited oxygen availability. Survival to 45 minutes and beyond suggests an open air pocket exists (i.e. patent airways 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 (106). Less than 20 % survive for more than two hours (107). Climate and topography affect the survival from avalanche burial (104). 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 chest outside of the snow) compared to 51.3 % of fully buried casualties (i.e. head and chest below the snow) die (108). An avalanche airbag reduces the risk of full burial and may also reduce burial depth. The reduction in mortality is less when the airbag does not inflate: in one study, non-inflation occurred in 20 % of use, 60 % of which was attributed to a deployment failure by the user (109). With risk of avalanches, winter sport athletes who are outside protected slopes should be equipped with, and trained in the use of, an avalanche airbag, an avalanche transceiver, a probe, and a metal-headed shovel. 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 analogue avalanche transceivers, attending avalanche rescue courses on correct tracking and probing, as well as regular training in deploying an avalanche airbag and working in coordinated groups will allow faster extrication. In mountain regions close to urban centers, helicopter rescue has revolutionized avalanche rescue because of fast transport times. Still, professional rescue should not be expected on scene within the first 20 minutes. 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 (79, 96, 97). In normothermic 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), outcome is substantially better (79, 110, 111).

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 2- and 8-fold greater than grass. In a snowy environment at 2000 m, UV-exposure is doubled (112, 113).

'Snow blindness' results from acute UV ocular injury. The degree of conjunctival and corneal injury ('ultraviolet keratitis') depends on energy intensity and exposure duration. Limbic injury causes pain when trying to focus the lens. Symptoms begin 4-10 hours after exposure and range from a 'gritty' sensation to severe 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 worsened by contact lens use (113, 114). A fluorescein eye stain test can locate corneal injuries.

Prevention

Sunglasses or goggles with side-protection, that 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 gently through the closed eyelid (113). In a next step use oral analgesics (e.g. paracetamol 1g $4X \cdot d^{-1}$). If available, administer a topical lubricant (e.g. antioxidant artificial tears), and apply

non-steroidal anti-inflammatory eye-drops (e.g. diclofenac 0.1 %). Topical anesthetics slow corneal recovery and should not be used outside emergency situations (e.g. descent from high-altitude). Consider topical antibiotics in severe cases to prevent infection. Cycloplegic eye-drops (cyclopentolate 1 %) may relieve pain but impair vision (113, 114).

Cold Water

The great cooling power of water means that some of the responses described for cold air (e.g. hypothermia) also occur in cold water, but sooner. Humans cool 4-5 times faster in cold water than in air at the same temperature (115).

There are four stages of immersion in cold water, each associated with specific hazards and each related to the cooling of different body tissues. Rapid skin cooling on initial immersion stimulates the cutaneous cold receptors; their dynamic response elicits the “Cold Shock”, a response not evoked by the slower rates of skin cooling in air. Cold shock is regarded as the most dangerous response caused by cold water immersion and affects men and women to a similar extent (116, 117). The greatest responses are observed at water temperatures between 10-15 °C (50-59 °F) for lightly clothed (swim-suited) individuals and include: a “gasp” response, hyperventilation, hypertension and increased cardiac workload (118). These can be precursors to drowning and cardiovascular issues. The initial gasp response is 2-3 liters (119, 120), larger than the lethal volume of sea water for drowning (1.5 L, (121)). The cold shock response peaks in the first 30 seconds of immersion and usually abates over the next 90 seconds as the peripheral cold receptors adapt to the new skin temperature. There is a significant possibility of aspirating water during

initial immersion when conscious control of breathing has been lost. In situations where the face is also immersed on initial immersion, co-activation of sympathetic and parasympathetic inputs to the heart can produce “Autonomic Conflict,” resulting in potentially fatal arrhythmias in a variety of sporting situations (e.g. open water swimming, triathlon, (122)).

After skin, the next body tissues to be affected by cooling are the superficial nerves and muscle. The arms are particularly susceptible to cooling due to their anthropometry (thin cylinders), anatomy (superficial nerves and muscles) and physiology (reliance on blood flow for warming) (100). Within about 20 minutes of immersion in water at about 12 °C (54 °F) peripheral neuromuscular cooling can significantly impair manual dexterity, grip strength and co-ordination, impacting swimming ability (100, 123).

Approximately 60 % of cold-water immersion deaths occur within the first minutes of immersion, long before hypothermia occurs (122). Under normal circumstances, adult humans will not become hypothermic in under 30 minutes in even the coldest water.

Prevention/Protection/Treatment

The cold shock response demonstrates temporal summation, with a greater response being observed with faster rates of entry into cold water (124). Those entering cold water are advised to rest with their airway clear of the water until they have regained control of their breathing (“float first”). Prolonged head-in breath holding should be avoided on initial immersion. For open cold water swimming events, swimmers should adapt to the water just before commencing the swim (or have in-water starts); this reduces the chance of aspirating water 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 habituation, with as few as six 2-min head-out cold water immersions over 3 days reducing the cold shock response by 50 % by the last immersion (125). A reduction of 25 % is still apparent 14 months later (126). Although habituation will reduce the ventilatory response on immersion, this may not translate to an improved swimming capacity (127).

Importantly, repeated immersion in cold water does not protect against neuromuscular incapacitation from peripheral cooling; protection for this response can only be achieved by limiting exposure or technological protection with protective clothing. In terms of swim failure, it appears that the upper arms (triceps region) is the most important region to protect (123, 128). 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 (129). Most sports persons use wet rather than dry suits. The primary determinants of the protection provided by a wet suit are its fit and thickness. The fit should be as tight as possible commensurate with sporting performance and the thickness varies between the 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) in wetsuits and 16 °C (61 °F) without wetsuits (130, 131). It is recommended that water temperatures below 18 °C (64 °F) are too cold for elite marathon swim racing without wet suit protection (132). 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) (133, 134).

Protection can also be afforded by appropriate event organization. Swimming events, including mass starts, can be organized and supervised to minimize the

chances of cardiac and respiratory-related problems (135). 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 (123).

Basic life support compressions and ventilations (2 rescue breaths then cardiopulmonary resuscitation at 30:2 ratio) should be used and for drowning victims, high concentrations of inspired oxygen given as soon as possible. All those suspected of aspirating water should be evacuated to hospital. For hypothermic casualties, follow the advice given in the section on “Accidental Hypothermia”.

Cold & Performance

Cold air and water exposure can potentially have deleterious effects on aerobic and strength performance (136-142). Interested readers are referred to previous reviews of the impact of cold exposure on exercise performance and the physiological mechanisms responsible for cold-related changes in performance (100, 143, 144).

Decreased muscle temperatures (T_{muscle}) lower VO_{2max}, exercise time, and power/sprint ability. For every 1 °C fall in muscle temperature there is a 4-6 % decline in these markers of performance (145-147). For example, an 8 °C decrease in T_{muscle} decreases power output by 31 % and maximal voluntary contraction (MVC) by 19 % (148). Low T_{muscle} (28-29 °C) also cause higher muscle lactate levels in both Type 1 and Type 2 muscle fibers (149, 150), and overall, blood lactate levels increase during exercise to a greater extent in cold compared to temperate environments (136, 151-156) suggesting a greater reliance on anaerobic metabolism. Maximal heart rate is lower by 10-30 beats·min^{-1} when T_{deep} is lowered

by 0.5 to 2.0 °C (157) and cold water decreases leg muscle blood flow during exercise (152, 158).

In cold air where there is little change in T_{deep} or T_{muscle}, there is a lack of consensus on whether aerobic performance declines: studies have demonstrated impairment (137-140), improvement (159, 160), or no change (161, 162). Two studies have examined, systematically, whether air temperature affects performance. Cycling time to exhaustion at 70 % VO_{2max} was longest at an ambient temperature (T_{amb}) of 11 °C while wearing shorts/t-shirt (137) with decrements seen at 4 °C and 31 °C. While wearing cross-country ski uniforms, Sandsund et al. (139) observed maximal running performance at T_{amb} between -4 °C (24.8 °F) and 1 °C (33.8 °F), with performance 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 ski uniform 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 exposure, this could reduce performance due to increased energy demands caused by heavy clothing, resistance to movement, and other equipment (4, 163). Upper-body performance in cross-country skiers is also reduced at colder T_{amb} (164, 165). Cold temperatures also can impact biomechanics and gait, increasing the energy demands of exercise (166). Furthermore, athletes need to be cognizant of terrain factors (i.e., ice, snow cover) that can cause slipping and coordination issues.

Combined Stressors

In the natural world it is rare for stressors such as heat and humidity, cold and altitude and cold and hyperbaric stress not to be combined or experienced sequentially. However, largely due to the way subject areas are organized, the

impact of combined stressors on human responses has received much less attention than the impact of isolated stressors. Between 1948 and 2012 there were only fourteen studies examining these areas with human participants (167). Since 2012, the number of studies has increased dramatically and the importance of this area of research for human performance, health and disease is beginning to be realized (168). The areas of combined stressors includes not only the beneficial or detrimental effects of combined stressors such as cold and hypoxia, it also includes cross-adaptation and cross tolerance between such stressors (Figure 3), at both the systemic and cellular levels[1]

Focusing on the combination of cold and altitude (hypoxia) (169), cold-induced thermogenesis is reduced in hypoxia (170), and T_{deep} falls faster (171). During cooling and warming the vasoconstrictor response can occur earlier and be released later if hypoxia is combined with cold, thereby increasing the "dose" of cold experienced and the likelihood of cold injuries (171, 172), although this remains a matter for debate (173).

Adaptation to the initial responses to cold water immersion *reduces* the subsequent responses to a hypoxic exposure (174) including: plasma epinephrine and norepinephrine, sympathetic nervous system response, heart rate, ventilation, oxygen consumption and carbon dioxide production, as well as symptoms of hypoxia and their severity (174). The proposed molecular targets for this cross adaptation

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

have included cold-inducible RNA-binding protein, heat shock protein 72 and 90α, putative RNA-binding protein 3 and the Sirtuin family of signaling proteins (175).

In contrast to the clear interaction between cold and hypoxia, it has been reported that cold acclimation does not alter the physiological or perceptual responses during subsequent exercise in the heat (176). The area of combined stressors is clearly fertile ground for further investigation with regard to the avoidance of injuries in the cold.

Conclusion

Coaches, athletes, medical personnel and officials need to understand the physiology, pathophysiology, prevention, protection and treatment of cold-related impacts on performance. From this understanding come optimal interventions for the maintenance of performance and avoidance of frostbite, non-freezing cold injuries, hypothermia, drowning and other medical events.

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Table 1. Factors predisposing to frostbite and non-freezing cold injury.

Climate	Individual Characteristics and Physiology
Wind	
Wetness, immersion in cold water	African-American or Afro-Caribbean ethnic background
Contact with cold materials	Male
High altitude	Children
Hypoxia	Elderly
Long duration or high amount of cold exposure	Previous cold injury
	Poor CIVD Response
	Homeless
Behavioral	**Individual/clinical**
Smoking	Coronary artery disease/ischemic heart disease, cardiac insufficiency, stroke
Alcohol use	Peripheral vascular disease
Drug use	Peripheral neuropathy
Inappropriate or constrictive clothing	Cold sensitivity
Prolonged stationary situation, immobility	Raynaud's phenomenon, white fingers
Fatigue, dehydration, malnutrition	Hand-arm-vibration syndrome, vibration
Use of emollients	Diabetes
Military rank/task	Hypothyroidism, hypopituitarism
Poor calorie intake	Depression, schizophrenia, dementia
	Neurovascular diseases
	Sweating or hyperhidrosis
	Previous cold injury
	Medication (vasoconstrictors)

References:
(7, 10-12, 16, 18, 21, 22, 25, 26, 31-34, 37, 39, 44-46, 53, 66, 75, 77, 177-191)

Table 2. Traditional historical classification of frostbite adapted from Fudge (192).

For field use, a simpler 2-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 (193).

Frostbite Degree	Physical Findings
1st Degree	Numbness, central white or yellow, waxy discoloration, surrounded by erythema and 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 hours, tissue loss involving entire thickness of skin
4th Degree	Injury is through the dermis, into subcutaneous tissue, muscle, and bone

Table 3. Signs and symptoms of hypothermia at different levels (194).

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 judgement	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 (195) and the Wilderness Medical Society classification (196). WMS - Wilderness Medical Society

Swiss system (195)			**WMS (196)**		
Category	**Clinical findings**	**Estimated core temperature (°C; °F)**	**Category**	**Clinical findings**	**Estimated core temperature (°C; °F)**
Stage 1	Clear consciousness with shivering	35-32 **°C** **95-89.6 °F**	**Mild**	Normal mental status, shivering, but not functioning normally/able to care for self	35-32 **°C** **95-89.6 °F**
Stage 2	Impaired consciousness without shivering	<32-28 **°C** **<89.6-82.4 °F**	**Moderate**	Abnormal mental status with shivering, or abnormal mental status without shivering, but conscious	32-28 **°C** **<89.6-82.4 °F**
Stage 3	Unconsciousness	<28-24 **°C** **<82.4-75.2 °F**	**Severe/profound**	Unconscious	<28 **°C** **<82.4 °F**
Stage 4	Apparent death	24-11.8 **°C** **75.2-53.2 °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. (197).

Table 5. Revised Swiss System for staging of accidental hypothermia (198). AVPU denotes Alert, Verbal, Painful and Unconscious, respectively.

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

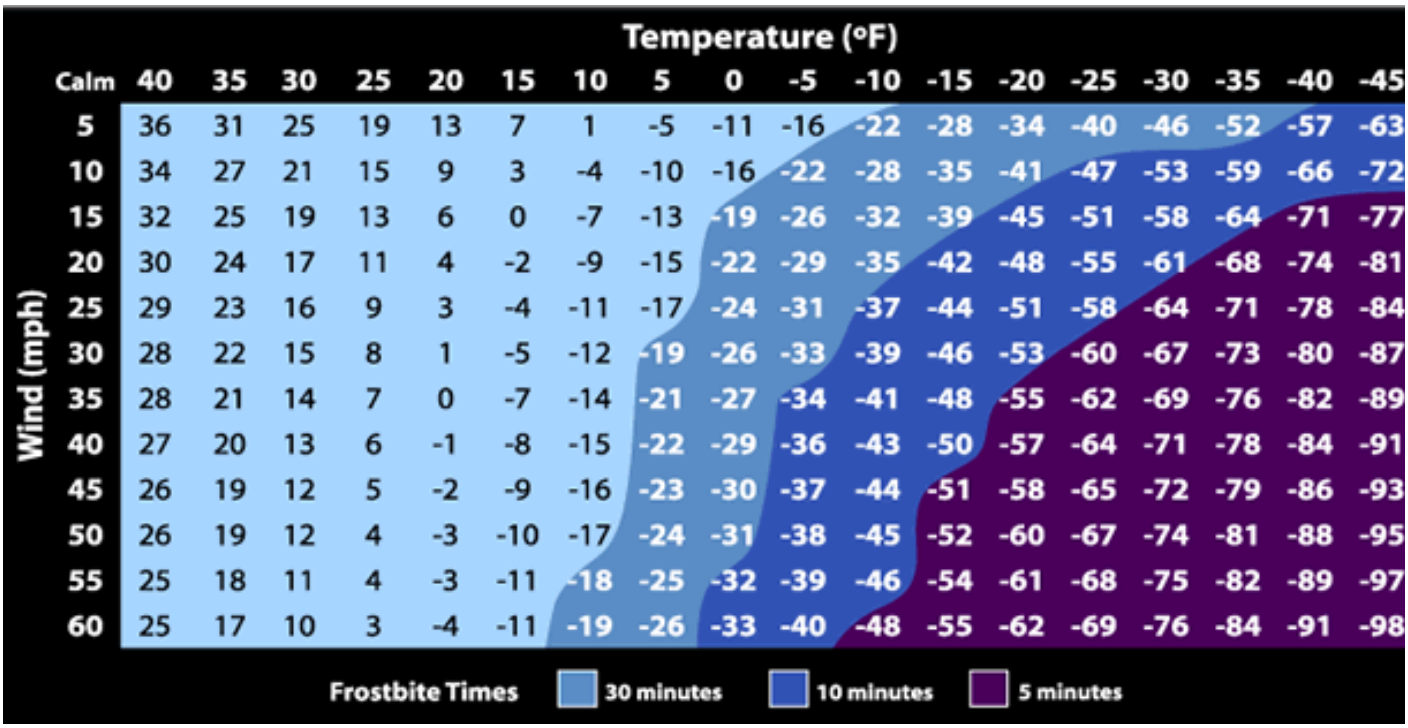
[a] In the Revised Swiss System, "Alert" corresponds to a GCS score of 15; "Verbal" corresponds to a GCS score of 9-14, including confused patients; "Painful" and "Unconscious" correspond to a GCS score <9. While shivering is not used as a stage-defining sign in the Revised Swiss System, its presence usually means that the temperature is >30°C (86 °F), a temperature at which hypothermic CA is unlikely to occur.[8]

[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 minute.[1]

[c] The transition of colours 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 1. Wind Chill Temperature 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 (40)

A.



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 2. Treatment algorithm for patients with accidental hypothermia, from (79). Definitions of parenthetical numbers are: (1) Decapitation; truncal transection; whole body decomposed or whole body frozen solid (chest wall not compressible); (2) SBP < 90 mmHg is a reasonable prehospital estimate of cardiocirculatory instability but for in-hospital decisions, the minimum sufficient circulation for a deeply hypothermic patient (e.g., <28 °C) has not been defined; (3) Swiss staging of accidental hypothermia; (4) Direct transport to an ECMO center is recommended in an arrested hypothermic patient. In remote areas, transport decisions should balance the risk of increased transport time with the potential benefit 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 (38-42 °C). In case of cardiac instability refractory to medical management, consider rewarming with ECLS; (6) If the decision is made to stop at an intermediate hospital to measure serum potassium, a hospital en route to an ECLS center should be chosen. HOPE and ICE scores should not be used in children; instead consider expert consultation. CPR denotes cardiopulmonary resuscitation, DNR, do-not-resuscitate, ECLS, extracorporeal life support, HT, hypothermia, MD, medical doctor, ROSC, return of spontaneous circulation, and SBP, systolic blood pressure.

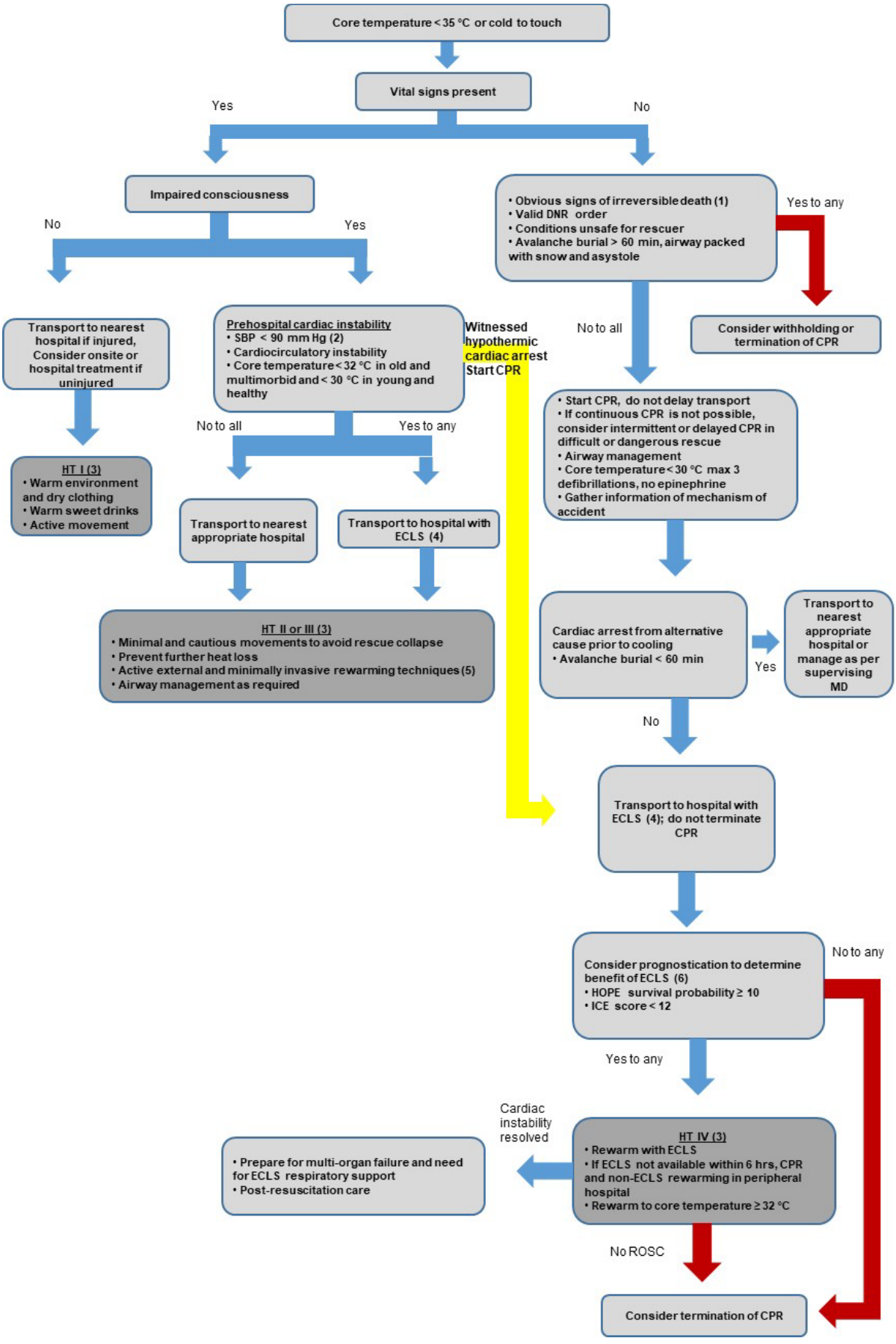

Core temperature < 35 °C or cold to touch
Vital signs present
Yes
No
Impaired consciousness
No
Yes
Transport to nearest hospital if injured, Consider onsite or hospital treatment if uninjured
HT I (3)
• Warm environment and dry clothing
• Warm sweet drinks
• Active movement
Prehospital cardiac instability
• SBP < 90 mm Hg (2)
• Cardiocirculatory instability
• Core temperature < 32 °C in old and multimorbid and < 30 °C in young and healthy
No to all
Yes to any
Transport to nearest appropriate hospital
Transport to hospital with ECLS (4)
HT II or III (3)
• Minimal and cautious movements to avoid rescue collapse
• Prevent further heat loss
• Active external and minimally invasive rewarming techniques (5)
• Airway management as required
Witnessed hypothermic cardiac arrest
Start CPR
• Obvious signs of irreversible death (1)
• Valid DNR order
• Conditions unsafe for rescuer
• Avalanche burial > 60 min, airway packed with snow and asystole
Yes to any
Consider withholding or termination of CPR
No to all
• Start CPR, do not delay transport
• If continuous CPR is not possible, consider intermittent or delayed CPR in difficult or dangerous rescue
• Airway management
• Core temperature < 30 °C max 3 defibrillations, no epinephrine
• Gather information of mechanism of accident
Cardiac arrest from alternative cause prior to cooling
• Avalanche burial < 60 min
Yes
Transport to nearest appropriate hospital or manage as per supervising MD
No
Transport to hospital with ECLS (4); do not terminate CPR
Consider prognostication to determine benefit of ECLS (6)
• HOPE survival probability ≥ 10
• ICE score < 12
No to any
Yes to any
Cardiac instability resolved
HT IV (3)
• Rewarm with ECLS
• If ECLS not available within 6 hrs, CPR and non-ECLS rewarming in peripheral hospital
• Rewarm to core temperature ≥ 32 °C
• Prepare for multi-organ failure and need for ECLS respiratory support
• Post-resuscitation care
No ROSC
Consider termination of CPR

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 pathways involved in cellular tolerance, or "Specific" involving a more specific response to a stimulus such as shivering or vasoconstriction.