

Ministry of Defence

Synopsis of Causation

Cold Injury

Author: Dr Adrian Roberts, Medical Author, Medical Text, Edinburgh
Validator: Dr Howard Oakley, Institute of Naval Medicine, Alverstoke, Gosport, Hampshire

September 2008

Disclaimer

This synopsis has been completed by medical practitioners. It is based on a literature search at the standard of a textbook of medicine and generalist review articles. It is not intended to be a meta-analysis of the literature on the condition specified.

Every effort has been taken to ensure that the information contained in the synopsis is accurate and consistent with current knowledge and practice and to do this the synopsis has been subject to an external validation process by consultants in a relevant specialty nominated by the Royal Society of Medicine.

The Ministry of Defence accepts full responsibility for the contents of this synopsis, and for any claims for loss, damage or injury arising from the use of this synopsis by the Ministry of Defence.

1. Definition

- 1.1. Cold injury is a term that encompasses both **systemic hypothermia** and **localised injury to a body part**. Systemic and local injury can present separately or in combination. This synopsis focuses primarily on localised cold injury. Systemic hypothermia is considered briefly in section 7. Cold urticaria, cold-induced [paraesthesia](#), [Raynaud's phenomenon](#), and cold-induced asthma are all related to cold exposure. However, these conditions are not customarily described as “cold injuries” and are not covered in this synopsis.
- 1.2. **Localised cold injury** covers a spectrum of damage that ranges from reversible changes to severe tissue destruction and loss. A clear distinction may be drawn between **freezing cold injury (FCI)**, where tissues become frozen below a temperature of -0.55°C , and **non-freezing cold injury (NFCI)**, which occurs when tissues are subjected to prolonged cooling that is insufficient to cause freezing. Freezing and non-freezing cold injury may coexist in a single individual or limb, although the dominant form of injury will usually be apparent. **Cold sensitivity** is a common sequela of cold injury.¹
- 1.3. **Non-freezing cold injury (NFCI)**
 - 1.3.1. **Chilblains** (synonym: **pernio**) represent a chronic [vasculitis](#) of the [dermis](#). The condition is provoked by repeated exposure to the cold, which causes constriction of the small arteries and veins in the skin. Subsequent re-warming results in leakage from the blood vessels into the tissues, which in turn causes inflammation and swelling of the skin. Chilblains characteristically develop several hours after exposure to cold but non-freezing temperatures in the presence of high humidity. Chilblains are less common in countries where the cold is more extreme because the air is less humid and people have adapted their living conditions and clothing. Chilblains are typically self-limiting.
 - 1.3.2. The terms “**trench foot**” and “**cold immersion foot (or hand)**” were coined to describe injuries sustained in wet conditions at non-freezing temperatures. Trench foot has a particular connotation with the trench warfare of World War I but remains a significant medical problem in military operations performed in cold weather. The condition has featured in many campaigns including World War II, the Korean War, and the Falklands conflict. Soldiers and sailors can be exposed to risk. It rarely presents in the civilian population.
 - 1.3.3. Trench foot follows protracted immersion in cold water, usually at 0°C to $+10^{\circ}\text{C}$, which gives rise to prolonged peripheral [vasoconstriction](#). Resultant [ischaemia](#) and changes in cell function damage the blood vessels, nerves, skin, and muscle. Clinically, NFCI appears to belong to a family of disorders in which there are different combinations of [ischaemia](#) followed by [hyperaemia](#) and subsequent sensitisation to temperature disturbance. The terms “**tropical immersion foot**” and “**paddy foot**” have been applied to injuries sustained by military personnel exposed to prolonged wetness in much warmer water. Another condition with similarities to NFCI is “**shelter limb**”, which affected civilians who were forced to take shelter in the deep tunnels of the London Underground during the blitz in World War II.^{1,2}

1.4. Freezing cold injury (FCI)

- 1.4.1. **Frostnip** is the mildest form of peripheral freezing cold injury. It is defined as freezing cold injury that, within 30 minutes of starting rewarming, recovers fully, leaving no residual symptoms or signs. A second episode of frostnip occurring during the same winter and in the same limb as a first episode is regarded as superficial frostbite, rather than recurrent frostnip.^{3,4}
 - 1.4.2. **Frostbite** usually affects the extremities and occurs as a consequence of acute freezing of tissues with microvascular occlusion and subsequent tissue [anoxia](#). The condition may be classified as superficial or deep. Frostbite can occur within minutes when skin is exposed to extremely low temperatures. Severe frostbite can have serious consequences, which may include loss of digits and limbs.
 - 1.4.3. **Contact frostbite** is a special type of freezing cold injury. Skin or [mucosal](#) contact with a supercold object, liquid (e.g. gasoline) or gas (e.g. evaporating liquid nitrogen) cools the contact area so rapidly, that there is no time for the normal vasoconstrictive cold response. The skin freezes with crystallisation of the intracellular fluid, and the cells die immediately. **Cold contact adhesion** causes an erosion or ulcer on forcible separation.⁵
- 1.5. In a historical context, cold injuries have been recognised predominantly as a military problem. Some of the earliest descriptions came from Baron de Larray, Napoleon's chief surgeon. In World War II, it was estimated that more than 7 million soldier fighting days were lost by Allied forces as a result of cold injury.⁶ As recently as the Falklands campaign of 1982, both British and Argentinean troops reported cold injuries after exposure to both freezing and non-freezing temperatures.² Cold injury is not limited to ground troops; high-altitude frostbite was first described in aviators in 1943. In fact, heavy bomber crews sustained more injuries from frostbite than from all other causes combined.
- 1.6. In cold climates such as Norway and Finland, local cold injuries remain a common problem of military operations and training in wintertime, especially during land manoeuvres.⁷ However in the past quarter century, the population distribution of cold injury has been seen to change with an increased incidence occurring in civilians. Those affected include the homeless and urban poor, wilderness enthusiasts including mountaineers, and people who engage in winter sports activities. Furthermore, cold injuries including frostbite are not confined to people who live in countries with an extremely cold climate. These conditions can affect people within the UK, predominantly in association with winter sports, rough sleeping, psychiatric illness, and misuse of drugs and alcohol.

2. Clinical Features

- 2.1. **Chilblains** consist of red-purple, itchy or tender lesions that appear as papules, macules, plaques, or nodules. They develop over a few hours and generally subside over the next 1-3 weeks. Occasionally, they progress to chronic pernio, which can persist for months, and may feature blistering, ulceration, scabs, scarring, or atrophy.⁸
 - 2.1.1. Chilblains present typically with a bilateral, symmetrical distribution. Common sites are the backs and sides of the fingers and toes, heels, shins, thighs (especially in horse riders), nose, and ears.
 - 2.1.2. Children and the elderly are most often affected. Children often experience recurrences each winter for a few years but an eventual complete recovery is usual. Chilblains in elderly people tend to get worse every year unless precipitating factors are avoided.
- 2.2. The common pathway for **trench foot** and the related conditions that are grouped under the title of **NFCI** involves exposure of one or more limbs to environmental conditions that result in reduced blood flow. This is followed by a period of reperfusion, which is accompanied by an acute syndrome of hyperaemia, swelling and pain, and which may in turn be followed by a chronic disorder.⁹ Although almost all cases of NFCI involve the feet, injured hands may also feature in as many as 25% of cases.¹⁰
- 2.3. Four distinct stages of NFCI are recognised.^{1,11,12}
 - 2.3.1. **During cold exposure.** [Vasoconstriction](#) causes the affected tissue to become cold and numb. Sensory changes range from local anaesthesia to loss of [proprioception](#). As a result, patients may report disturbance of gait, clumsiness, and stumbling. The extremities may initially be a bright red colour, but later change to a paler colour, even completely blanched white. Pain and swelling do not feature at this stage.
 - 2.3.2. **Following cold exposure.** The second stage appears with warming and is fleeting in nature. The extremities characteristically change colour from white to mottled pale blue whilst remaining cold and numb. Initial swelling may develop and peripheral pulses are often impalpable.
 - 2.3.3. **Hyperaemia.** The hyperaemic stage typically lasts from 2 weeks to 3 months. The affected extremity becomes swollen with hot, red, and dry skin. The microcirculation is sluggish, although the peripheral pulses become full and bounding. Anaesthesia is replaced by [paraesthesiae](#) and pain, which may be intense and disturb sleep. Pain is typically most severe in the sole of the foot. Blistering may occur and, in very severe cases, areas of skin may start to show signs of nonviability before becoming overtly gangrenous in the next stage.
 - 2.3.4. **Following hyperaemia.** The final stage of NFCI may last for weeks to years and, for some patients, persists for the remainder of their lives. Obvious physical signs are absent. Inflammatory responses are usually reduced and limb temperature falls. Sequelae may develop as described at section 5.3.3.

- 2.4. **Frostnip** is most likely to be experienced by skiers and others who participate in outdoor winter sports, affecting the nose, ears, or tips of digits. It presents with initial pain and blanching, with subsequent numbness of the affected body part. Tissue injury does not occur, provided that progression to frostbite and multiple exposures are avoided.
- 2.5. In **frostbite**, two distinct **mechanisms** are believed to be responsible for tissue injury:
- (i) **Initial freeze injury.** Direct injury causes cellular death at the time of exposure.
 - (ii) **Reperfusion injury occurring with rewarming.** Progressive microvascular [thrombosis](#) causes advancing injury in otherwise viable cells and leads to deterioration, [necrosis](#) and gangrene.
- 2.5.1. Freezing injuries that have become partially defrosted and then refrozen are responsible for the most recalcitrant injuries because such “freeze–thaw–refreeze” cycles result in progressively severe thrombosis and blood vessel damage.¹³
- 2.5.2. The extremities are most susceptible to frostbite. Hands and feet account for over 90% of all recorded injuries. Other sites include ears, nose, cheeks, and penis.² In a Canadian study, anatomic distribution was 19% upper extremity, 47% lower extremity, 31% combined upper and lower extremity, and 3% facial or trunk only.¹⁴ Other studies have reported somewhat different distributions. German studies from World War II suggest that facial injuries are more common than the above figures would indicate, but that they generally receive less medical attention because they are functionally less significant.
- 2.5.3. Frostbite injuries vary markedly in severity although, at the time of initial evaluation, most frostbite injuries appear similar. For this reason, the following classification into four degrees is applied after rewarming:
- **First degree frostbite.** Numb central white plaque with surrounding erythema but no blistering
 - **Second degree frostbite.** Blister formation surrounded by erythema and oedema. The blisters fill with clear or milky fluid in the first 24 hours
 - **Third degree frostbite.** Death of skin and subcutaneous tissues forming haemorrhagic blisters that result in a hard black [eschar](#) 2-3 weeks later
 - **Fourth degree frostbite.** Tissue necrosis, gangrene, and eventual full-thickness tissue loss; initially the affected body part nearly always appears hard, cold, white, and anaesthetic

These categories have not proven useful in predicting the extent of damage after rewarming. Some authors prefer to describe just two classes of injury, **superficial** (corresponding to 1° and 2° - confined to skin and subcutaneous tissues) and **deep** (3° and 4° - also involving muscles, bones, and joints). These may predict outcome more accurately.²

- 2.5.4. Several imaging techniques have been used in frostbite with a view to assessing tissue viability. Technetium-99m pertechnetate bone scanning ([scintigraphy](#)) has become the standard imaging study used within the first few days after injury and magnetic resonance imaging (MRI) has shown some promise in the

early determination of eventual bone and soft tissue viability. However, no prognostic technique has as yet proved sufficiently accurate as to support a case for early surgical intervention.¹⁵

3. Aetiology - Weather Conditions and Heat Loss

- 3.1. The severity of cold injury depends on a variety of environmental and situational factors including the temperature, duration of exposure, wind speed, solar conditions, type of activity that is undertaken during exposure, and amount of protective clothing that is worn.
- 3.2. Body temperature may fall as a result of heat loss by radiation, evaporation, conduction, and convection. Under normal conditions, around 60% of heat is lost by radiation. However, heat loss from convection and conduction can increase dramatically in cold, wet, and windy environments. Hypothermia develops faster with submersion in water than with exposure to cold air.
- 3.3. Convection plays an important role in the aetiology of FCI. The rate of heat transfer by convection increases significantly with increasing air velocity. Thus, given equal air temperatures, an object cools more quickly under windy conditions than in a calm environment. However, it must be appreciated that higher wind speeds do not alter the actual air temperature, nor can they cause an exposed object to become colder than the ambient air.
 - 3.3.1 When the wind blows, it disperses the boundary layer, which is a thin insulating layer of air that is warmed up by the body and lies close to the skin. If this protective layer keeps getting blown away, skin temperature will drop and the person will feel colder. Wind also causes evaporation of moisture on the skin, drawing more heat away from the body. Wet skin loses heat much faster than dry skin.
 - 3.3.2 **Wind chill** is a concept that relates the **rate** of heat loss under windy conditions to an equivalent air temperature for calm conditions. Following criticism of previous methods of measuring wind chill, a revised formula has been adopted by the Joint Action Group for Temperature Indices (JAG/TI), a group drawn from representatives of US federal agencies and Canadian national ministries.¹⁶ The UK Met Office has now adopted the JAG/TI algorithm.
 - 3.3.3 The JAG/TI algorithm provides a wind chill temperature index (WCTI) based on heat loss from the face. The WCTI factors the air temperature and wind speed to provide a temperature-like number that represents the air temperature that, under calm conditions, would produce the same cooling effect as the actual air temperature produces in windy conditions. For example, a wind of 10 km/hr, measured at the standard anemometer height of 10 metres, will make an air temperature of -5°C feel the same as -9°C feels in calm air, and is therefore described as a wind chill of -9 . If the air temperature remains stable at -5°C but the wind increases to 25 km/hr, the wind chill falls to -12 ; at a wind speed of 50 km/hr, the wind chill reaches -15 .
 - 3.3.4 Frostbite cannot occur unless the actual air temperature is below freezing and, in practice, well below the established -0.55°C freezing point of skin. This finding has been attributed to the protection afforded to the skin by cold-induced [vasodilatation](#) (CIVD), which produces cyclic rewarming as the skin cools in ambient temperatures that are below freezing point. For an acute exposure in an average individual, the air temperature generally needs to fall to

–10°C to –15°C or lower for the risk of frostbite to be imminent. Less extreme temperatures can cause frostbite during longer exposures, perhaps because of the ultimate reduction in CIVD.¹⁷ However, CIVD is a contentious issue and it has been suggested that it is often absent in military situations.

- 3.4. The longer the exposure and/or the colder the apparent temperature (wind chill), the deeper the tissue damage will become in frostbite. The risk of finger frostbite is low at air temperatures that are above -10°C, irrespective of wind speed, whilst below -25°C there is a pronounced risk even at low wind speeds.³ The JAG/TI has published data that allows their wind chill index to be correlated to estimates of time to frostbite. However this remains open to dispute and is not in accord with UK armed forces experience. In a study of Royal Marine personnel conducted in 1986, most FCI occurred at temperatures above -30°C with the highest risk area lying between -9°C and -19°C. Nearly two-thirds of all injuries occurred within a wind chill risk zone that was considered to be relatively “safe” as one wherein cold injuries were unlikely to occur.¹⁸
- 3.5. It has been suggested that inhabitants of cold regions e.g. Inuits may “feel the cold” less than people who dwell in a temperate climate. However, there is no evidence that it is possible to become acclimatised to cold weather. Furthermore, any person who has a history of previous cold-related injury or illness, however mild, retains an increased risk of renewed incidence.

4. Aetiology – Predisposing Factors

4.1. Risk factors for **chilblains** include:

- Season: characteristically occur during autumn and winter and resolve completely when the weather becomes warmer
- Increased prevalence of chilblains in young, thin females (generally under the age of 30 years)
- Familial tendency
- Peripheral vascular disease due to diabetes, smoking, hyperlipidaemia
- Poor nutrition e.g. anorexia nervosa
- Connective tissue disease e.g. systemic lupus erythematosus, [Raynaud's phenomenon](#), scleroderma
- [Acrocyanosis](#) or [erythrocyanosis](#)
- Bone marrow disorders

Conversely, chilblains may improve during pregnancy

4.2. **NFCI (trench foot)** follows exposure to wet, cold conditions with ambient temperatures above freezing. A typical duration of exposure is often quoted as 1-2 days, but NFCI may result from exposures of less than 1 hour (e.g. immersion injury in very cold water) to up to a week under less severely cold conditions.^{11,13} The severity of trench foot is determined by the degree of cold, wetness of the tissue, the duration of exposure, and individual variability.

4.2.1. Most authorities believe that NFCI is a vascular [neuropathy](#) and that intense and prolonged cold-induced peripheral [vasoconstriction](#) is the most significant feature in the aetiology of the condition.

4.2.2. A variety of factors may arise during military campaigns that, in combination, predispose susceptible combatants to NFCI. These include constrictive clothing and footwear, lack of protective equipment, lack of nutritious food, dehydration, fatigue, restricted mobility, and a cold, wet, inhospitable environment.

4.2.3. The prevalence of NFCI appears to be higher in military personnel operating in combat situations than in civilians or non-combat military personnel exposed to similar or even more severe environmental conditions. Studies suggest that combat stress is a significant contributing factor.¹¹

4.2.4. There are individual differences in the susceptibility to cold injury that are related to an individual's vascular reactions to the cold environment. Given the same environmental conditions, individuals capable of maintaining higher local blood flow and skin temperatures are less susceptible to NFCI than are those with lower peripheral blood flow and skin temperatures. Individuals who have had prior cold injuries face an increased risk of NFCI if re-exposed, especially if there is evidence that they suffer from residual cold sensitisation.

4.3. The risk of **frostbite** is related to environmental conditions including ambient temperature and wind chill effect, as described in section 3. Individual sensitivity to

cold is important. An increased risk is manifested in individuals who are unwell, unfit, hungry, or who have a history of previous cold-related injury or illness, however mild.

4.3.1. Predisposing factors for frostbite may be classified as those that increase heat loss, decrease heat production, decrease the insulation of the clothing, make people especially susceptible to cold, or cause them to behave inappropriately. Factors that affect susceptibility to frostbite include the following:¹⁹

- Direct exposure of skin and poor insulation of the clothing: insulation can be insufficient when clothing is wet, tight-fitting, permeable to wind, or does not cover the cold-sensitive body parts
- Transport in an open vehicle
- Homelessness, especially rough sleeping
- Trauma and assuming a cramped position for a prolonged period of time: both may result in inability to move the extremities and lead to soft tissue swelling, impaired circulation, and a consequent increased incidence of cold injury
- Fatigue and poor physical fitness
- Dehydration
- Hands and feet that sweat easily
- Previous history of cold weather injury
- Alcohol consumption has been identified as a particular risk factor because of its effects on an individual's judgement as well as its vasodilatory effects
- Major psychiatric disorder may lead to inappropriate behaviour or clothing
- Circulatory impairment e.g. [atherosclerosis](#). The vasoconstrictive effects of smoking also contribute to increased risk
- Treatment with beta-blockers, which causes constriction of the blood vessels, resulting in colder hands and feet
- Diabetes mellitus: the mechanisms that are likely to be involved are peripheral vascular disease and peripheral neuropathy
- Individuals who suffer from [Raynaud's phenomenon](#) and hand-arm vibration syndrome appear to have an increased risk of frostbite injuries to the fingers. This factor is particularly relevant for reindeer herders, who are exposed to vibration through gripping the handlebars of their snowmobiles^{20,21,22}

4.4. Ethnicity is also important. Individuals of Black Caribbean and Black African ethnic origin have a significantly increased susceptibility to localised cold injury that extends to both NFCI and FCI. NFCI that affects the hands in addition to the feet is particularly common in these ethnic groups.¹⁰ US studies have reported an approximate four-fold increased risk of cold injury for African-American male soldiers as compared to their White male counterparts.^{23,24}

5. Prognosis

- 5.1. **Chilblains** generally respond poorly to treatment. The main focus is on preventative measures including suitable indoor heating, warm outdoor clothing, and the avoidance of smoking. Conservative treatment includes elevation and application of moisturising lotions. A topical steroid cream may provide symptomatic relief of itching. Treatment that promotes peripheral vasodilatation e.g. nifedipine has met with some success.²⁵ Although acute chilblains are usually self-limiting without long-term sequelae, the pain from chronic pernio injury can last a lifetime, especially when the condition commences during childhood.¹²
- 5.2. FCI and NFCI can generally be prevented with proper preparation for outdoor activities in a cold environment. Preventive behaviour depends on awareness of the risks and the early symptoms and signs of cold injury. Key preventive measures focus on hydration, nutrition, shelter, and suitable protective clothing. **Cold sensitisation** and **chronic pain** are important sequelae of both FCI and NFCI, and these complications are analysed in detail in section 6.
- 5.3. Preventative measures for **NFCI (trench foot)** include education and training, frequent foot-care routines, protective non-constrictive clothing and footwear, avoidance of immobility, and rotation of troops between exposed frontline positions and the rearguard wherever possible.
 - 5.3.1. Initial treatment of trench foot consists of immediate removal from the cold, wet environment and slow rewarming. Rapid rewarming of NFCI exacerbates the injury.¹¹ Once trench foot is established, treatment is essentially palliative, including analgesia, protection of pressure spots, and local and systemic measures to combat inflammation and infection. Moist liquefaction gangrene can occur and sequential surgical amputations may be necessary over a period of weeks.⁶
 - 5.3.2. Conventional analgesics are usually ineffective, and amitriptyline is the drug of choice for the treatment of pain following NFCI. Administration of amitriptyline should commence as soon as pain is felt, as there is evidence that early treatment may reduce the incidence of later intractable pain.⁴
 - 5.3.3. With regard to sequelae, increased sensitivity to cold stimuli is a very common finding (see section 6). [Hyperhydrosis](#) occurs in a significant but smaller proportion of cases, and may predispose to recurrent fungal infection. Small areas of numbness or [paraesthesia](#) may remain in perpetuity, although more substantial sensory loss is unusual. Other complications that may occur include deep aching and pain on pressure, flexion contractures with claw deformities, shedding of the nail, disturbances of nail growth, muscle atrophy, fallen arches, osteoporosis, and chronic ulceration.^{1,6,12}
- 5.4. **Frostnip** is reversible. Warming of the cold tissue results in return of sensation and function without tissue loss.
- 5.5. Various preventative measures can be taken to prevent **frostbite**. However, once it has occurred, initial **management** for all classes of frostbite is the same.

- 5.5.1. Rapid rewarming is the first treatment objective. Rapid rewarming reverses the direct effects of ice crystal formation within the tissue, but does nothing to prevent the progressive dermal [ischaemia](#) seen in the post-thaw phase. Repeated bouts of thawing and refreezing result in worsening injury. Tissue should not be rewarmed in the field if there is a risk of refreezing.
- 5.5.2. Post-thaw care is largely supportive, focusing on measures to provide adequate analgesia, promote optimal functional recovery and prevent further injury while awaiting the demarcation of irreversible tissue destruction. Smoking is contraindicated and weight bearing should be avoided until complete resolution of oedema. Daily hydrotherapy aids [debridement](#) of devitalised tissue and maintains the active and passive ranges of movement essential for the preservation of function. Normal sensation will not return for several weeks, until tissue healing is complete.
- 5.5.3. The natural history of a full-thickness frostbite injury involves the gradual demarcation of the injured area, with dry gangrene or mummification clearly delineating the nonviable tissue. Differentiation between viable and nonviable tissue does not begin until a minimum of 2 to 5 days after the exposure. There is often a discrepancy between the limit of the skin lesions and the extent of damage to deeper structures. Surgery is not normally contemplated until demarcation is complete, aiming to tidy up the consequences of auto-amputation. Consequently, operations are usually delayed until at least 6 months after initial injury. Premature surgical intervention risks insult to potentially viable, recovering tissue and a consequent increase in tissue loss.
- 5.5.4. Early non-surgical intervention is critical in terms of ultimate outcome. The degree of irreversible damage is related to the length of time that the tissue remains frozen. Delay in seeking medical care for more than 24 hours is associated with an 85% likelihood that surgical intervention will be required. Patients who present within the first 24 hours require surgery in less than 30% of cases. Often, the permanent tissue loss is much less than originally suspected. In an Alaskan series, only 10.5% of patients required amputation, usually involving only [phalanges](#) or portions of phalanges.
- 5.6. Recovery from frostbite may be affected by complications.
 - 5.6.1. Complications that arise during the early stages include tetanus (prophylaxis is given where indicated), dehydration due to cold diuresis, and [rhabdomyolysis](#) with attendant risk of subsequent renal failure.
 - 5.6.2. Infection is the most important early complication; it has been reported in 13% of urban frostbite victims and is a predictor of poor outcome.²⁶
 - 5.6.3. Frostbitten tissues seldom recover completely and long-term sequelae may be evident. A trend is observed whereby the severity of sequelae appears most pronounced in third and fourth degree injuries. However, some individuals with first degree injury may suffer from significant sequelae, whilst others with third degree injury may exhibit a more benign clinical course, being able to return to cold exposure after healing is complete.²⁷

- 5.6.4. The more common sequelae of frostbite may include cold sensitisation (see section 6), pain, skin colour changes, [hyperhidrosis](#), and [hyperkeratosis](#). Disturbances of sensation may occur, including [hyperaesthesia](#), decreased sensitivity of touch, or numbness.^{13,28}
- 5.6.5. Other long-term sequelae include premature closure of epiphyses in children, decreased nail and hair growth, tremor, neuropathy, muscle atrophy, tuftal resorption of terminal [phalanges](#), osteoporosis, and contractures. Chronic ulcers and scars that break down repeatedly are not uncommon, especially when there has been infection in the injured tissues. Squamous cell carcinomas of low malignancy may arise in the ulcers left at the site of previous frostbite.¹
- 5.6.6. Very rarely, severe frostbite exposure leads to localised changes that mimic osteoarthritis, typically affecting the interphalangeal joints of the hand. Sparing of the thumb is characteristic, although not invariable, an observation that may arise as a result of the tendency to clasp the thumb within a clenched fist during cold exposure. The timing is variable and the onset of symptoms may occur between a few months and 10 years or more after the injury. Plain radiographs reveal an erosive osteoarthritis with [subchondral osteosclerosis](#) and, unusually for osteoarthritis, large cystic defects.²⁹

6. Cold Sensitisation and Chronic Pain

- 6.1. Cold sensitisation and chronic pain are common sequelae of FCI and NFCI. Presentations may be mixed, or may appear at either extreme. Chronic pain is the dominant symptom in less than 5% of patients, and is a neuropathic pain that can resemble reflex sympathetic dystrophy or a complex regional pain syndrome.
- 6.2. **Cold sensitisation** is reported in 60-80% of cases and may present during the resolution of the [hyperaemia](#) associated with NFCI or about 1 month following FCI. The feet are most commonly affected, hands and male external genitalia may also be involved, but the face and ears are usually unaffected. The relative distribution of cases of cold sensitisation between FCI and NFCI, and between severe and mild cold injury, remains uncertain. However, the condition appears to be most common following mild and even subclinical cases of NFCI affecting the feet. Many of the British servicemen who experienced mild NFCI in the Falklands War did not report sick during the conflict. However, on their return to the UK, most were markedly cold sensitised.^{1,11} Often, individuals who sustained even mild degrees of cold injury remain symptomatic after many years.³⁰ Cumulative cold injuries may result in very severe cold sensitisation.³
- 6.3. Although results from a formal trial are awaited, foot spa rewarming, 20-30 minutes at least once per day in water at 40°C, appears to have shown very promising results in alleviating symptoms.³ Otherwise, with the exception of avoiding further cold exposure, there has been no evidence that any intervention alters the course of cold sensitisation to a significant extent. There appears to be considerable variation in the clinical course. Some patients recover completely, some remain unchanged indefinitely, and some deteriorate although rarely to the point whereby tissue viability is compromised. Changes including complete resolution may still occur many years after the initial injury. It is generally held that the more severe the condition, the less the likelihood is of complete recovery. Emigration or even temporary residence in a hot climate has been associated with complete recovery.
- 6.4. **Chronic pain** has been reported in more than 70% of cases of NFCI and may be the dominant symptom.¹¹ The pain, which is often triggered by cold exposure and associated with vasoconstriction, may become intractable and has been likened to post-herpetic neuralgia. Conventional analgesics, narcotics and non-steroidal anti-inflammatory drugs are unhelpful. Amitriptyline may be beneficial, with the addition of pregabalin should pain continue to break through. Gabapentin is not normally of benefit and many patients never achieve any useful pain relief. Sympathectomy (either by means of temporary blocks or permanent surgical methods) should be avoided, as experience has shown that it often results in medium- and long-term deterioration.^{3,10}

7. Hypothermia

- 7.1. Systemic **hypothermia** is defined as a fall in core body temperature to 35°C or less. In more general terms, it can be regarded as a decrease in body temperature that renders the body unable to generate adequate heat to continue its natural functions.¹³
- 7.1.1. The condition is classified as either primary or secondary. Primary, accidental hypothermia occurs in healthy individuals who are exposed to severe cooling, usually as a result of prolonged environmental exposure or cold water immersion. In secondary hypothermia, the individual is predisposed to developing the condition by a separate illness that affects heat production or thermoregulation. In these circumstances, hypothermia can develop in the presence of mild cold stress.
- 7.1.2. Hypothermia is further classified as;
- mild (core temperature 35° to 32.2°C);
 - moderate (core temperature <32.2° to 28°C); and
 - severe (core temperature <28°C)
- 7.1.3. With the introduction of aggressive resuscitation measures, patients have been revived from core temperatures as low as 14-15°C.
- 7.2. The [hypothalamus](#) is responsible for regulating the human body's temperature and the physiological response to cold. Physiological adaptations to heat loss, including shivering and increased muscle tone, remain intact in cases of mild hypothermia. Early symptoms can be vague, including nausea, vomiting, fatigue, and dizziness. Early signs feature tremulousness, acceleration of the heart and respiratory rates, and profound [vasoconstriction](#). In severe hypothermia, shivering is abolished, metabolism decreases, and heat loss is accepted passively. Severe hypothermia is characterised by loss of reflexes, coma, and cardiac arrhythmia. Some patients who could yet be revived may appear dead with pupillary dilatation, unobtainable pulse and apparent rigor mortis.^{6,13}
- 7.3. As the body's core temperature falls, effects are spread across multiple organ systems as follows:^{6,13}
- 7.3.1. **Cardiovascular.** Initially heart rate, blood pressure, and cardiac output increase. Subsequently, after a plateau phase, these same parameters commence a progressive and often rapid decline, which may progress to shock. Changes in the electrocardiogram may develop including, at temperatures below 32°C, a distinctive J wave (secondary wave following the QRS complex). As temperature falls below 30°C, the [myocardium](#) becomes irritable, leading to arrhythmias including atrial fibrillation and ventricular fibrillation. [Asystole](#) occurs at temperatures below 25°C.
- 7.3.2. **Neurological.** Initially, there is a loss of fine motor skills, manifest as clumsiness or lack of coordination. This is followed by a progressive decrease in cognitive performance and loss of deep tendon reflexes, with [dysarthria](#), [ataxia](#), and loss of gross motor skills. Consciousness is usually lost between 31° and 27°C.

- 7.3.3. **Renal.** Initially, urinary output is often maintained because of impaired renal tubular sodium and water reabsorption and inhibition of antidiuretic hormone (cold diuresis). Eventually renal blood flow decreases as the cardiac output declines, ultimately leading to acute renal failure.
- 7.3.4. **Respiratory.** Respiratory drive is increased in the early stages but progressive respiratory depression occurs below 33°C. Occasionally, hypothermia results in the production of a large amount of mucus (cold bronchorrhoea). Depression of ciliary action and the cough reflex predispose to [atelectasis](#) and aspiration.
- 7.3.5. **Gastrointestinal.** [Ileus](#), bowel wall oedema, depressed hepatic drug detoxification, gastric erosions, and haemorrhagic pancreatitis may all occur.
- 7.3.6. **Metabolic.** Hyperglycaemia is relatively common because insulin release and insulin uptake by membrane receptors is inhibited at temperatures below 30°C. Variable serum electrolyte disturbances may occur.
- 7.3.7. **Haematological.** Reduced platelet function and prolongation of clot formation may occur (particularly relevant in trauma patients with hypothermia).
- 7.3.8. **Musculoskeletal.** [Ischaemia](#) may lead to [rhabdomyolysis](#) with the attendant risk of subsequent renal failure.
- 7.4. The aetiology of **primary hypothermia** is principally associated with accidental environmental exposure in severe weather conditions. Exhaustion acts as an exacerbating factor.
- 7.4.1. Hypothermia is more common at the extremes of age. Young children have an increased surface area to body mass ratio, which increases the rate of heat loss. The elderly often have a decreased ability to sense cold and a decreased capacity for metabolic heat production and [vasoconstriction](#).
- 7.4.2. Every hypothermic patient requires an extensive and thorough survey to determine the presence of conditions that have led to or resulted from the hypothermia.¹³ Specific conditions that may lead to **secondary hypothermia** include:⁸
- Malnutrition (reduces metabolic heat production)
 - Psychiatric illness
 - Mental impairment
 - Recreational and therapeutic drugs, including alcohol, barbiturates, phenothiazines, morphine, and clonidine
 - Suicide attempt
 - Central nervous system dysfunction that can affect the [hypothalamic](#) thermoregulatory centre, including degeneration, trauma, or neoplasm
 - Spinal cord damage, peripheral neuropathy, and autonomic neuropathy. People with diabetes mellitus may develop peripheral or autonomic neuropathy
 - Endocrine conditions including hypothyroidism, adrenal insufficiency, and hypoglycaemia
 - Dermal dysfunction such as burns and [erythroderma](#)

- Falls and trauma, including head injury or fracture causing immobility
- Systemic infection is common and can either be a precipitant or a complication of hypothermia

7.5. Many cases of hypothermia could be prevented with proper preparation for outdoor activities in a cold environment. The patient who has become hypothermic is treated by rewarming with close monitoring of heart rhythm, pulse and blood pressure. Depending on the severity of hypothermia, the available techniques involve:

- Passive external rewarming (i.e. spontaneous rewarming following removal from the hypothermic environment)
- Active external rewarming (e.g. heating blankets, heating lights)
- Active core rewarming (e.g. heated intravenous fluids, body cavity [lavage](#) with warmed solutions, heated and watersaturated inhaled air, and extracorporeal circulatory rewarming by means of cardiopulmonary bypass)

7.5.1. Generally, mortality from hypothermia is high, particularly when the condition is moderate or severe, although most survivors of hypothermia do not experience long-term sequelae. A multicentre study of patients with accidental hypothermia reported 17% mortality, with 85% of the fatalities having presented with a core temperature less than or equal to 32.2°C.³¹ Other studies have reported mortality rates as high as 80%, primarily when hypothermia has been caused by infection or underlying illness. Hypothermia in trauma patients is also associated with a particularly high mortality rate.

7.5.2. Mortality from severe hypothermia (core temperature, <28°C) is especially high. Even with resort to cardiopulmonary bypass (the resuscitative method of choice for patients with cardiac arrest or cardiovascular instability and core temperatures of less than 32.2°C) mortality reaches 40-50%.^{32,33} However, when this treatment is successful, it is encouraging to note that the long-term outlook appears favourable. A study has been published of the late outcome for survivors who had been rewarmed with cardiopulmonary bypass for severe hypothermia with cardiac arrest. The subjects were all previously healthy young patients, most of whom had been involved in mountaineering accidents or suicide attempts. Severe neurological and other medical problems were evident in the early period after rewarming, and extensive rehabilitation was often necessary. Nevertheless, at follow-up after an average of 6.7 years, hypothermia-related neurological or neuropsychological deficits were either absent or mild with patients able to resume their former activities and lifestyles.³⁴

8. Summary

- 8.1. Cold injury is a collective term that encompasses both systemic hypothermia and localised injury to a body part. Localised cold injury covers a spectrum of damage that ranges from reversible changes to severe tissue destruction and loss. A clear distinction may be drawn between freezing cold injury and non-freezing cold injury,
- 8.2. Historically, cold injuries were recognised predominantly as a military problem and they remain a significant medical problem in military operations performed in cold weather. In recent years, the incidence within the civilian population has increased.
- 8.3. Chilblains are caused by a chronic vasculitis and are provoked by repeated exposure to cold but non-freezing temperatures in the presence of high humidity. They are generally self-limiting without long-term sequelae, although the condition can occasionally progress to a chronic form.
- 8.4. NFCI (trench foot and related conditions) results from extended exposure to wet conditions at non-freezing temperatures, which gives rise to prolonged peripheral vasoconstriction that damages blood vessels, nerves, skin, and muscle. Combat stress appears to be a significant contributing factor. Once established, treatment is essentially palliative.
- 8.5. Frostnip is the mildest form of peripheral cold injury. It is reversible with rewarming and consequently does not ordinarily result in any loss of tissue or function.
- 8.6. Frostbite causes tissue damage by initial freeze injury and ischaemia that occurs on rewarming. Classification into superficial and deep frostbite provides an indication of the prognosis. Surgery, where required, is ordinarily delayed until at least 6 months from the date of injury.
- 8.7. Long-term sequelae, especially cold sensitisation and chronic pain, are common following both FCI and NFCI. Cold sensitisation, whereby the affected extremity tends to become very cold and remain so for prolonged periods, is reported in 60-80% of cases following cold injury. There is considerable variation in the clinical course. Cold sensitisation predisposes to further cold injury.
- 8.8. Systemic hypothermia is defined as a fall in core body temperature to 35°C or less. Hypothermia may occur in a healthy individual who is exposed to severe cooling (primary) or in a person who is predisposed to developing the condition by a separate illness (secondary). The effects of hypothermia are spread across multiple organ systems. Mortality is high, although with recent therapeutic advances, patients have been revived from core temperatures as low as 14-15°C. Late outcome appears to be favourable for survivors of primary hypothermia who were previously healthy.
- 8.9. Hypothermia and localised cold injuries are largely preventable with proper preparation for outdoor activities in a cold environment.

9. Related Synopses

10. Glossary

acrocyanosis	Condition, more common in women than men, characterised by persistent discolouration of the skin of the extremities with sweating and coldness of the digits. Caused by constriction of the small blood vessels in the limbs.
aesthesia	Perception by the senses. Hence: <i>hyperaesthesia</i> , increased sensitivity to sensory stimuli; <i>paraesthesia</i> , abnormal sensation such as burning or prickling.
anoxia	Lack of oxygen supply to the tissues.
asystole	Cardiac standstill or arrest, absence of heartbeat.
ataxia	Failure of co-ordinated muscle movements.
atelectasis	Partial or complete collapse of the lung, usually due to obstruction of a bronchus.
atherosclerosis	Progressive narrowing and hardening of arteries over time.
debridement	Removal of dead, infected, or foreign material from a wound.
dermis	Connective tissue layer of skin, underlying the epithelium (surface layer).
dysarthria	A problem with speech due to difficulty with articulation.
endothelium	The layer of cells lining the cavity of blood vessels.
erythrocyanosis	Condition, more common in women than men, characterised by swelling and a reddish discolouration of the limbs in response to cold.
erythroderma	Widespread reddening of the skin due to dilatation of the blood vessels.
eschar	A dry, inelastic, often constricting scab, which is produced, for example, by a burn.
hyperaemia	Excess of blood in a part. Hence: <i>hyperaemic</i> .
hyperhidrosis	Excessive perspiration.
hyperkeratosis	Overgrowth of the corneous layer of the skin.
hypothalamus	A region of the brain involved in the regulation of glands, water balance, blood sugar, fat metabolism, and body temperature. Hence: <i>hypothalamic</i> .

ileus	Obstruction of the intestines.
ischaemia	Decreased flow of oxygenated blood to a part of the body.
lavage	Washing out.
mucosa	Membrane that lines a body cavity. Hence: <i>mucosal</i> .
myocardium	The muscular layer of the heart wall.
necrosis	Changes indicative of cell death.
neuropathy	A functional disturbance and/or pathological change in the peripheral nervous system.
osteosclerosis	Abnormal increase in density of bone – opposite of osteoporosis.
phalanges	The bones of the fingers and toes (plural of phalanx).
proprioception	The mechanism involved in the self-regulation of posture and movement.
Raynaud's phenomenon	Intermittent bilateral attacks of ischaemia of the fingers and toes (sometimes also ears and nose) characterised by severe pallor, often with accompanying pain and pins and needles. May be of unknown cause, when it is termed Raynaud's disease, or associated with an underlying condition such as an autoimmune disorder.
rhabdomyolysis	Destruction of skeletal muscle cells.
scintigraphy	Radioisotope scan that involves the administration of an appropriate radionuclide followed by a scan to record the distribution of radioactivity.
sepsis	The presence of organisms in the blood.
subchondral	Beneath the cartilage.
thrombosis	The development of obstruction due to blood clot formation (thrombus) within a blood channel.
vasculitis	Inflammation of a vessel, predominantly a blood or lymph vessel.
vasoconstriction	A decrease in calibre of the blood vessels, leading to reduced blood flow to a part.
vasodilatation	An increase in calibre of the blood vessels.

11. References

- ¹ Francis TJR, Oakley EHN. Cold injury. In: Tooke JE, Lowe GD, editors. A textbook of vascular medicine. London: Hodder Arnold; 1996. p. 353-70.
- ² Murphy JV, Banwell PE, Roberts AH, McGrouther DA. Frostbite: pathogenesis and treatment. *J Trauma* 2000;48(1):171-8.
- ³ Army Medical Directorate. Climatic injuries in the armed forces: prevention and treatment. Joint Services Publication 539. London: Ministry of Defence; 2003.
- ⁴ Oakley EHN. Proposed treatment protocols for cold injuries. INM report no. 2000.042. Gosport, UK: Institute of Naval Medicine; 2000.
- ⁵ Lehmuskallio E, Hassi J, Kettunen P. The skin in the cold. *Int J Circumpolar Health* 2002 ;61(3):277-86.
- ⁶ Jurkovich GJ. Environmental cold-induced injury. *Surg Clin North Am* 2007;87(1):247-67, viii.
- ⁷ Lehmuskallio E, Lindholm H, Koskenvuo K et al. Frostbite of the face and ears: epidemiological study of risk factors in Finnish conscripts. *BMJ* 1995;311(7021):1661-3.
- ⁸ Biem J, Koehncke N, Classen D, Dosman J. Out of the cold: management of hypothermia and frostbite. *CMAJ* 2003;168(3):305-11.
- ⁹ Oakley EHN. A review of the treatment of cold injury. INM report no. 2000.026. Gosport, UK: Institute of Naval Medicine; 2000.
- ¹⁰ Imray CH, Oakley EHN. Cold still kills: cold-related illnesses in military practice freezing and non-freezing cold injury. *J R Army Med Corps* 2005;151(4):218-22.
- ¹¹ Thomas JR, Oakley EHN. Nonfreezing cold injury. In: Pandolf KB, Burr RE, editors. Medical aspects of harsh environments, volume 1. Washington, DC: Borden Institute; 2001. p. 467-90.
- ¹² Hamlet M. Peripheral cold injury. In: Holmér I, Kuklane K editors. Problems with cold work: proceedings from an international symposium held in Stockholm, Sweden, Grand Hôtel Saltsjöbaden, November 16–20, 1997. Solna, Sweden: Arbetslivsinstitutet; 1998. p. 127-31.
- ¹³ Ulrich AS, Rathlev NK. Hypothermia and localized cold injuries. *Emerg Med Clin North Am* 2004;22(2):281-98.
- ¹⁴ Valnicek SM, Chasmar LR, Clapson JB. Frostbite in the prairies: a 12-year review. *Plast Reconstr Surg* 1993;92(4):633-41.
- ¹⁵ Bhatnagar A, Sarker BB, Sawroop K et al. Diagnosis, characterisation and evaluation of treatment response of frostbite using pertechnetate scintigraphy: a prospective study. *Eur J Nucl Med Mol Imaging* 2002;29(2):170–5.
- ¹⁶ US Department of Commerce: Office of the Federal Coordinator for Meteorological Services and Supporting Research. Report on wind chill temperature and extreme heat indices: evaluation and improvement projects. [Online]. 2003 Jan [cited 2007 Feb 13]. Available from: URL:http://www.ofcm.gov/jagti/r19-ti-plan/pdf/entire_r19_ti.pdf
- ¹⁷ Wilson O, Goldman RF. Role of air temperature and wind in the time necessary for a finger to freeze. *J Appl Physiol* 1970;29(5):658-64.
- ¹⁸ Wagstaff MA, Pethybridge RJ. Cold injuries: Norwegian winter deployment 1986. INM report no. 9/87. Gosport, UK: Institute of Naval Medicine; 1987.
- ¹⁹ Rintamaki H. Predisposing factors and prevention of frostbite. *Int J Circumpolar Health* 2000;59(2):114-21.
- ²⁰ Valter I, Maricq HR. Prevalence of Raynaud's phenomenon in 2 ethnic groups in the general population of Estonia. *J Rheumatol* 1998;25(4):697-702.
- ²¹ Virokannas H, Anttonen H. Risk of frostbite in vibration-induced white finger cases. *Arctic Med Res* 1993;52(2):69-72.
- ²² Ervasti O, Juopperi K, Kettunen P et al. The occurrence of frostbite and its risk factors in young men. *Int J Circumpolar Health* 2004;63(1):71-80.
- ²³ Candler WH, Ivey H. Cold weather injuries among U.S. soldiers in Alaska: a five-year review. *Mil Med* 1997;162(12):788-91.
- ²⁴ DeGroot DW, Castellani JW, Williams JO, Amoroso PJ. Epidemiology of U.S. Army cold weather injuries, 1980-1999. *Aviat Space Environ Med* 2003;74(5):564-70.
- ²⁵ Rustin MH, Newton JA, Smith NP, Dowd PM. The treatment of chilblains with nifedipine: the results of a pilot study, a double-blind placebo-controlled randomized study and a long-term open trial. *Br J Dermatol* 1989;120(2):267-75.
- ²⁶ Urschel JD. Frostbite: predisposing factors and predictors of poor outcome. *J Trauma* 1990;30(3):340-2.

-
- ²⁷ Rosen L, Eltvik L, Arvesen A, Stranden E. Local cold injuries sustained during military service in the Norwegian Army. *Arctic Med Res* 1991;50(4):159-65.
- ²⁸ Ervasti O, Hassi J, Rintamaki H et al. Sequelae of moderate finger frostbite as assessed by subjective sensations, clinical signs, and thermophysiological responses. *Int J Circumpolar Health* 2000;59(2):137-45.
- ²⁹ Kahn JE, Lidove O, Laredo JD, Blétry O. Frostbite arthritis. *Ann Rheum Dis* 2005;64(6):966-7.
- ³⁰ Oakley EHN. The long-term sequelae of cold injury among "The Chosin Few". INM report no. 96043. Gosport, UK: Institute of Naval Medicine; 1996.
- ³¹ Danzl DF, Pozos RS, Auerbach PS et al. Multicenter hypothermia survey. *Ann Emerg Med* 1987;16(9):1042-55.
- ³² Lazar HL. The treatment of hypothermia. *N Engl J Med* 1997;337:1545-7.
- ³³ Vretenar DF, Urschel JD, Parrott JC, Unruh HW. Cardiopulmonary bypass resuscitation for accidental hypothermia. *Ann Thorac Surg* 1994;58(3):895-8.
- ³⁴ Walpoth BH, Walpoth-Aslan BN, Mattle HP et al. Outcome of survivors of accidental deep hypothermia and circulatory arrest treated with extracorporeal blood warming. *N Engl J Med* 1997;337:1500-5.