Physical Causes: Heat, Cold, and Other Atmospheric Factors

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Contents

Abstract

Several ambient conditions or physical work-related exposures can induce skin changes. These may either represent normal reactions to an "abnormal" level of an exogenous factor, e.g., high or low temperature, or abnormal reactions to conditions which are normally compensated by skin homoeostasis and thus generally tolerated. Heat induces skin damage directly in

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terms of thermal or electrical burns of various degrees and extent, requiring adequate emergency treatment. However, heat may be applied to skin also via infra-red radiation or prolonged direct exposure to moderate heat, causing Erythema ab igne. Ambient heat may cause sweat retention in different layers of the eccrine sweat glands and thereby miliaria. Similar conditions predispose to intertrigo, a macerated, Erythematous eruption in body folds, especially with excessive sweating and in obese persons. Cold has considerable, potentially life-threatening systemic effects (exposure) and can cause frostbite. Moderate cold may cause an abnormal reaction in the susceptible in terms of perniosis (chilblains) of exposed acral body regions, mostly the fingers. Prolonged wetness, usually of the feet and lower legs, especially if combined with low temperature, can cause "immersion foot" as a nonfreezing cold injury. As another nonfreezing injury, and with mechanical effects of rowing contributing to etiology, "pulling boat hands" have been described. In addition to temperature, ambient humidity may have some adverse effect on skin, for instance, in terms of low outdoor humidity (typically less than 10 mg/l) contributing to skin irritation and eventually irritant contact dermatitis. Similarly, low indoor humidity, e.g., in clean-rooms, has been found to cause highly pruritic, if clinically largely inconspicuous skin eruptions in a large share of exposed workers. Symptoms may be aggravated by, e.g., fine, irritating particles.

Keywords

Ambient humidity · Burn · Clean room · Cold · Dermatitis · Electrical burn · Erythema ab igne · Heat · Immersion foot · Miliaria · Squamous cell carcinoma · Sweat retention · Temperature

1 Core Message

- Heat induces skin damage not only directly in terms of thermal or electrical burns, but also via infra-red radiation or prolonged direct exposure to moderate heat (Erythema ab igne).
- Sweat retention in different layers of the eccrine sweat glands may cause miliaria.
- Cold has considerable systemic effects and can cause frostbite or, if only moderate, perniosis (chilblains).
- Prolonged wetness of the feet and lower legs, especially if combined with low temperature, can cause "immersion foot" as a nonfreezing cold injury.
- Both low outdoor and (subsequent) low indoor air humidity may cause skin irritation and favor irritation by other agents, e.g., wet work.

2 Introduction

Injuries by some of the physical factors mentioned in this chapter are among the most ancient health risks of mankind and, indeed, of all living objects. Other factors have been introduced by man. Partly, these produce the same morbidity, such as Erythema ab igne not caused by open fires, from caves to castles, but by holding lap top computers on the thighs for prolonged periods. However, in a few instances, man-made physical factors may produce novel skin changes, such as thermal effects of microwave radiation, or low-humidity dermatoses in "clean rooms." The following chapter will present the most important types of injury in a systematic way.

3 Heat

3.1 Thermal Burns

Burns result from exposure to extremes of heat. The lowest temperature at which a burn can occur has been estimated to be 44 °C (111 °F) (Moritz and Henriques 1947). Burns may be of industrial, domestic, or environmental origin. Industrial burns are common (Cason 1981) and may have characteristic occupational patterns (Renz and Sherman 1994; Woods et al. 1996). They may be caused by direct contact with hot objects, radiation heat (infra-red, IR), or hot steam or air, e.g., emitted by improperly handled heat torches. Moreover, accidental exposure to laser energy may cause thermal destruction through absorption

Table 1 Classification of burns based on the depth of the burns (From Burke and Bondoc 1993)

Classification	Surface appearance	Sensation	Outcome/prognosis
First-degree	Dry, erythematous, no blisters	Painful, hypersensitive	Complete healing within 1 week; no scar
Second-degree (superficial)	Blisters, red oozing base, good capillary refill, blanching on pressure	Painful, extremely sensitive to pinprick	Complete healing within 3 weeks; may be erythematous early after healing
Second-degree (deep)	Blisters may be present; pale, indurated areas; some areas red	Some insensitive areas; many areas anesthetic to pinprick	Firm, thick scar with loss of hair follicles, sweat glands, and skin pigmentation; healing may take 1 month
Third-degree	Pearly white or brown opaque gray; firm leathery, dry	No sensation	Total skin loss includes all appendages; heals by scar formation if small

by skin chromophores such as melanin and hemoglobin, well known from the therapeutic application of different lasers in dermatology. Classification of burns is based on the depth of the burn as first, second, or third degree (Table 1; Burke and Bondoc 1993).

Although in many countries dermatologists are often not involved in the acute-phase treatment (Jeschke 2016) of more severe burns, they frequently are requested to evaluate impairment of patients with healed burns. Pigmentation, vascularity, pliability, and scar height are the main parameters (Sullivan et al. 1990) for dermatological evaluation. Sometimes scarring is extensive enough to limit joint mobility or muscle strength (Jonsson et al. 1997) in addition to producing varying degrees of disfigurement. Squamous cell carcinoma arising from burn scars was described by Marjolin in 1828; the frequency of this complication is unknown. The criteria established by Ewing (1935) for associating a carcinoma with a previous burn are still considered valid.

Burns may cause either hyperpigmentation or hypopigmentation. When total loss of pigment occurs, it denotes deeper burns with destruction of melanocytes, and recovery of skin color is doubtful. With more superficial burns, hyperpigmentation may result, depending on the depth of the burn and the genetic background of the patient. Blacks and other dark skinned persons show the greatest hyperpigmentation, which on the face and other exposed areas may be very disfiguring. Fading occurs slowly; a final evaluation of permanency should not be made until 18–24 months after the burn has completely healed.

3.2 Electrical Burns

These occur from environmental, domestic, and industrial sources. Out of 290 fatal factory accidents in Great Britain, 21 were due to electric shock; a larger number died from burns after contact with domestic 240 V alternating (50 Hz) current (Cason 1981). The lesions are due to heat and direct injury by electricity, the severity depending, e.g., on current voltage, thickness, and wetness of the skin, and duration of contact (Kennedy 1992). High voltage burns are severe, while low voltage (<1000 Volt) burns are milder, but penetrate more deeply than is apparent following nerves and vessels, extending some distance away from the edge of the visible wound. Even 24 V may produce deep injury (Benmeir et al. 1993). Late rupture of blood vessels can occur even as long as 2 weeks after the electrical burn (Kennedy 1992). Electrothermal burns may be caused by touching a heated electrical element. Burns from cardiac defibrillators have caused local skin ulcers. The depth of electric flash burns depends on the temperature of the agent and the duration of contact. Campbell and coworkers (1996) reported high-voltage electrical injury in two hang-glider pilots from 11,000 V power lines. Magnetic resonance imaging (MRI) is a valuable diagnostic aid in the care of high-voltage electrical burns (Nettelblad et al. 1996).

Burns from lightning cause a bizarre, superficial Erythema which is considered pathognomonic (Bartholome et al. 1975). The skin shows numerous erythematous macules arranged in a streaked feather- or fern-like pattern. Blanching on diascopy does not occur, and in those persons who survive fading occurs in 24–48 h. The condition is probably due to the transmission of static electricity along the superficial vessels, similar to that occurring when an electrodessiccating current is used to destroy small angiomas. Lightning injuries are an occupational risk of outdoor workers such as railroad and highway construction workers, surveyors, geologists, foresters, and agricultural workers.

3.3 Erythema ab igne

Infrared radiation, composed of wavelengths of 800–170,000 nm, results in thermal burns at temperatures over 44 °C (Zalar and Harber 1985). Chronic exposure to heat can result in Erythema ab igne and skin cancer. It is characterized by a mottled, reticulate hyperemia with melanoderma and telangiectasia and sometimes superficial epidermal atrophy and subepidermal blistering. Erythema ab igne occurs after prolonged exposure to heat, i.e., infra-red radiation, which is usually insufficient to produce burn. At one time the condition was common on the anterior legs and inner thighs of women who sat for long periods before an open fire and on the abdomens of patients with chronic abdominal pain who applied heating pads for hours at a time. New cases are now being seen as heating with wood fires has become more common again. Heater cushions still cause Erythema ab igne (Dvoretzky and Silverman 1991). Erythema ab igne may be a sign leading one to suspect hypothyroidism (Bashir and Chew 2016). It has also been reported as a marker of chronic pancreatitis (Mok and Blumgart 1984).

Workers who may develop Erythema ab igne include stokers, blacksmiths, glassblowers, bakers (especially those using old-fashioned brick-lined ovens), and cooks and others working over a heat source. Historically, the condition in stenographers who sit very close to radiators in cold weather has been described (Schwartz et al. 1957). According to Bashir and Chew (2016), it also occurs in the face of silversmiths and jewelers and on the arms of foundry men. A more recent source of Erythema ab igne mediated by contact, and not radiation, is laptop computers, if held for many hours and days (several case reports published, e.g., Bachmeyer et al. 2009). In mentally disturbed patients with thermophilia, bizarre areas of Erythema ab igne have been encountered (Bashir and Chew 2016).

After an interval of up to over 30 years, thermal skin injury and Erythema ab igne may proceed to cancer (Kaplan 1987). Several variants, mostly depending on a certain geographical influence or habit, have been described (Kanerva 1999). Infrared radiation may significantly enhance aging and carcinogenic effects of ultraviolet radiation (Kligman and Kligman 1984).

3.4 Erythermalgia

Primary erythermalgia usually arises in childhood and is a rare autosomal dominant neuropathy characterized by the combination of recurrent burning pain, warmth, and redness of the extremities (Tang et al. 2015). Secondary erythermalgia develops in adult age in association with or without an apparent underlying disorder. It is characterized by bilateral symmetric burning and redness of the lower, or sometimes upper, extremities. Symptoms can be initiated by exercise or exposure to heat, while rest and cold briefing relief (Drenth and Michiels 1994; Gaur and Koroscil 2009). Thus, occupational factors may aggravate symptoms (Table 2). Avoidance of triggering situations by lifestyle modification (Gaur and Koroscil 2009) has given the best results, but treatment may be difficult, and symptoms may persist throughout life. Secondary erythermalgia can arise as a side effect of drugs but otherwise the etiology is yet unknown (Drenth and Michiels 1994; Drenth et al. 1997). The past 20 years have not produced literature on "secondary erythermalgia," but focused on the inherited, primary variant.

Table 2 Workers potentially exposed to excessive heat

Type of work
Animal rendering workers
Asphalt workers
Bakers
Bitumen workers
Boiler heaters
Cannery workers
Chemical plant operators working near hot containers and furnaces
Cleaners
Coke oven operators
Cooks and other kitchen workers
Firemen
Foundry workers
Glass manufacturing workers
Greenhouse workers
Kiln workers
Miners in deep mines
Outdoor workers during hot weather
Sailors passing hot climatic zones
Shipyard workers when cleaning cargo holds
Smelter workers
Steel and metal forges
Textile manufacturing workers (weaving, dyeing) maintenance workers in nuclear plants
Tight protective clothing (all occupations), e.g., maintenance worker in nuclear plants
Tire (rubber) manufacturing workers

3.5 Miliaria

Miliaria is caused by sweat retention. Heat causes swelling of the keratin within sweat ducts, resulting in poral closure and rupture of the ducts immediately beneath the obstruction. If the obstruction occurs within the stratum corneum, miliaria crystallina (sudamina) results. This produces small, clear vesicles that soon rupture and lead to desquamation. Cutaneous bacteria, particularly Staphylococci, seem to play a role in the pathogenesis of miliaria (Mowad et al. 1995). The skin surface may or may not be faintly Erythematous. It is most commonly seen after mild nonspecific damage to the epidermis and/or profuse sweating. While miliaria crystallina is usually asymptomatic, patients may become concerned when they suddenly notice the entire palm is desquamating (Lobitz 1962). Widespread involvement may in rare cases interfere with thermoregulation.

When closure occurs somewhat deeper in the epidermis, in the granular layer, firm vesicles are formed accompanied by marked pruritus. Called miliaria rubra, or prickly heat, it is easily confused with contact dermatitis. This condition is more troublesome than miliaria crystallina because the eruption may be quite extensive and accompanied by paroxysms of burning and itching. The lesions are small erythematous macules and vesicles unassociated with follicular openings; a hand lens aids in visualization. The lesions may appear a few days after exposure to hot, humid environment, but most commonly appear after one to several months (Lillywhite 1992).

The trunk and intertriginous areas are particularly involved, while the palms and soles are spared. When poral closure is severe, hyperpyrexia and heat exhaustion occur, causing a decrease in work efficiency. The lesions that later develop may be pustular from infiltration of inflammatory cells and may be complicated by secondary bacterial infection.

If obstruction is even deeper, i.e., in the dermoepidermal junction zone, miliaria profunda results (Kirk et al. 1996), producing deep-seated asymptomatic vesicles, that appear much like gooseflesh, but close examination shows that they spare the follicles. The lesions consist of pale white papules 1–3 mm in diameter, which are most prominent on the trunk. Erythema and pruritus are mild or absent. This serious condition is caused by widespread inactivation of sweat glands from prolonged exposure to a hot environment. It usually follows an extended period of miliaria rubra. Heat exhaustion and collapse are common sequelae. A condition called tropical anhidrotic asthenia with acute fatigue, nausea, dizziness, palpitations, tachycardia, and malaise has been observed among military personnel during wartime in very hot, humid environments. Tropical anhidrotic asthenia is secondary to widespread miliaria profunda in particularly adverse conditions (Cage et al. 1987).

Treatment of miliaria consists of cooling the patient to reduce sweating. Mild, nonocclusive

lotions may be used. In miliaria crystallina, removal of the damaged area by mechanical means or by natural sloughing stops the process. In miliaria rubra, a week of rest from the inciting factors allows removal of the damaged portion of the epidermis by natural desquamation while in miliaria profunda rest in cool surroundings for several weeks is needed for complete recovery. Systemic and topical steroids are not helpful.

Occupations where excessive heat exposure can occur are listed in Table 2; in addition, work in the tropics may be inadvisable for very susceptible persons.

3.6 Intertrigo

A macerated, erythematous eruption in body folds – intertrigo – can occur from excessive sweating, especially in obese persons. Friction between opposing surfaces in addition to sweat retention are important etiologic factors. Secondary bacterial or Candida infection commonly accompanies intertrigo. The groin, axillae, and interdigital areas are favored sites. In particular, the interdigital space between the third and fourth fingers is a common site for intertrigo and secondary Candida infection among cannery workers, bartenders, medical and dental personnel, and others performing wet work for prolonged periods. Cooks, swimming instructors, nurses, and others exposed to moisture are also disposed to this condition. An important differential diagnosis, if finger web spaces are affected, is interdigital dermatitis as a common, early type of irritant contact dermatitis in wet work occupations (Schwanitz and Uter 2000). This will, however, show scaling and probably fissuring without maceration and should respond well to emollient treatment and improved skin protection measures.

3.7 Miscellaneous Conditions

Acne vulgaris and rosacea can be aggravated by prolonged chronic exposure to heat, especially intense heat from ovens, steam, open furnaces, or heat torches. Herpes simplex may be triggered by sudden blasts of heat. Prolonged exposure to excessive heat also increases irritability, decreases workers' ability to concentrate, and results in a generally lower level of efficiency.

4 Cold

The effect of coldness is the result of a complex interaction of climatic factors (air temperature, mean radiant temperature, humidity, and wind), protection (clothing), and metabolic heat production (activity). The nature of cooling encompasses (i) whole-body cooling, (ii) extremity cooling, (iii) convective cooling (wind chill), (iv) conductive cooling (contact), and (v) airway cooling (Holmér 1993). Cooling the brain leads to confusion and later to impaired coordination, while cooling the limbs results in numbness and clumsiness, making the performance of intricate tasks difficult.

Local cooling in most cases produces discomfort and harmful effects, before more significant whole-body cooling develops. With strong wind or exposure to very low temperature, frostbite of unprotected skin may quickly develop (see below). However, as digit cooling largely depends on whole-body heat balance, it is important to control body cooling by selection and use of appropriate protective clothing (Holmér 1993).

Everybody reacts to cold, but in addition to physiologic responses to cold, abnormal reactions may occur in some individuals. Chilblains may result from chronic exposure to moderate cold. Cold plays an important role in, e.g., Raynaud's phenomenon, cryoglobulinemia, and cold urticaria. Cutaneous reactions to cold can be divided into reactions to abnormal cold and abnormal reactions to "normal" cold. The diseases caused or aggravated by cold are listed in Table 3.

4.1 Frostbite

There are three stages of freezing cold injury: (i) massive vasoconstriction causing a rapid fall in skin temperature, (ii) the hunting phenomenon, i.e., transient cyclic vasodilatation by the opening of arteriovenous anastomoses causing a cyclic rise and fall in skin temperature, and (iii) if cold

Table 3 Diseases caused or aggravated by cold

I. Reactions to abnormal cold
1. Cold injury
2. Frostbite
3. Nonfreezing cold injury
(a) Immersion or trench foot
(b) Immersion foot
(c) Tropical immersion foot
II. Abnormal reactions to cold
1. Perniosis
2. Pulling boat hands
3. Acrocyanosis
4. Erythrocyanosis
5. Livedo reticularis
6. Cold urticaria
7. Cold erythema
8. Cold panniculitis
9. Sclerema neonatorum
10. Subcutaneous fat necrosis of the newborn
11. Cryoglobulinemia
12. Raynaud's phenomenon

exposure continues, freezing, as the skin temperature falls to approach ambient temperature (Kulka 1965). Risk increases with alcohol use and smoking (Bashir and Chew 2016).

The events that follow freezing and nonfreezing cold injury are similar and include: (i) arterial and arteriolar vasoconstriction, (ii) excessive venular and capillary vasodilatation, (iii) increased endothelial leakage, (iv) erythrostasis, (v) arteriovenous shunting, (vi) segmental vascular necrosis, and (vii) massive thrombosis (Heller Page and Shear 1993).

The degree of cellular injury depends on (i) the minimum temperature, (ii) the duration of time at that temperature, (iii) the cooling rate, with rapid cooling causing more destructive intracellular ice crystal formation, and hence more destruction as is obvious from cryotherapy, and (iv) rewarming rate. In slow rewarming, intracellular ice crystals become larger and more lethal for the cell; (v) finally, repeated freeze–thaw cycles lead to greater injury. Different cell types vary in their susceptibility to cold. Melanocytes are very sensitive, and damage occurs at −4 °C to −7 °C (25–19 °F). Accordingly, cryotherapy causes hypopigmentation (Heller Page and Shear 1993).

Low air temperatures and high wind speeds ("wind chill") are associated with an increased risk of freezing of the exposed skin. As the skin surface temperature falls from −4.8 °C to −7.8 °C, the risk of frostbite increases from 5% to 95% (Danielsson 1996). Frostbite additionally causes impairment of circulation due to slowly progressing vasoconstriction.

In its mildest form, only redness and pain are present. In more severe cases, tissue destruction and blistering occur and this may be superficial, full thickness or involve deep tissues analogous to the burns (Table 4). In its most extreme form, gangrene and loss of limb may result. Exposed parts, i.e., toes, feet, fingers, ears, nose, and cheeks, are most often affected.

As tissue temperature falls, the area becomes numb, and the initial redness is gradually replaced by a white, waxy appearance with blistering and later necrosis; the affected part becomes deceptively anesthetic. In the early stages, it is difficult to predict the extent of tissue loss becoming perceivable after rewarming, especially concerning deeper structures like muscle and bone. Accurate estimation may clinically not be possible for several weeks (Knize et al. 1969); however, magnetic resonance imaging (MRI) and angiography (MRA) provide for early recognition of damaged tissue (Barker et al. 1997). The more superficial the injury, the better the prognosis, especially if infection is absent. However, even without loss of tissue, long-term effects may include telangiectasia (Huh et al. 1996), vasomotor instability with Raynaud-like changes, paresthesia, and hyperhidrosis. This is attributed to damage to the blood vessels and sympathetic nerves. Squamous cell carcinoma may arise in the old scars (Rossis et al. 1982).

In slow rewarming, intracellular ice crystals become larger and more lethal for the cells. Therefore, treatment entails rapidly rewarming the part for which a whirlpool or water bath or warm air for 20 min (until the most distal part is flushed) is useful. A temperature of 40–42 °C (104–107.6 °F) (Golant et al. 2008) has been recommended, e.g., employing warm baths for 15–30 min. Rewarming should not begin until definite medical care can be provided, to avoid repeated freeze–thaw cycles (Golant et al. 2008). After

Table 4 Four-degree classification of frostbite (Roberts 2008, after Heil et al. 2016)

Degree of frostbite	Appearance after rewarming
First degree	Numb central white plaque surrounded by erythema but no blistering
Second degree	Blister formation surrounded by erythema and edema
	Blisters filled with clear or milky fluid in first 24 h
Third degree	Death of skin and subcutaneous tissues forming hemorrhagic blisters resulting in black eschar 2–3 weeks later
Fourth degree	Tissue necrosis, gangrene and eventually full thickness tissue loss; initially body part is hard, cold, white, and numb post rewarming

Table 5 Workers potentially exposed to excessive cold

Cooling room workers
Divers
Dry ice workers
Firefighters
Ice makers
Liquefied gas workers
Outdoor workers during cold weather
Packing house workers
Refrigerated warehouse workers
Refrigeration workers
Winter sports instructors

rewarming, the affected part is rested at usual room temperature. Rewarming is painful and causes an increase in Erythema and blistering. The pain should be relieved with analgesics; nonsteroidal anti-inflammatory drugs (NSAIDs) also contribute to lessening levels of prostaglandins and thromboxanes contributing to vasoconstriction (Bashir and Chew 2016). The damaged part should be elevated and blisters left intact. Infection must be treated vigorously. As second-line treatment, the intra-arterial infusion of tissue plasminogen activator (tPA) or administration of acetylsalicylic acid with iloprost have been shown to reduce the rate of amputations in third-degree frostbite (Bashir and Chew 2016). The popular old idea of rubbing the affected part with snow has an adverse, even disastrous effect.

Historically, the persons at greatest risk of frostbite have been military personnel. Today, many cases are associated with alcohol consumption, homelessness in urban centers, and car breakdown (Miller and Chasmar 1980). Frostbite also occurs in winter sports, e.g., cross country skiers or backpackers. Occupations at risk include oil pipeline workers in northern regions, utility maintenance personnel, sailors, especially those working on icebreakers, fishermen, firefighters, mail delivery persons, rescue personnel, researchers in cold laboratories and polar areas, and others who work outdoors in cold regions (Table 5).

4.2 Immersion Foot/Nonfreezing Cold Injury

Formerly called trench foot, immersion foot results from exposure to cold temperatures above freezing for several days. In the presence of moisture and constrictive clothing, however, continuous exposure for as little as 19 h may be sufficient (Rietschel and Allen 1976). Immersion foot is less severe than frostbite and develops in three stages: initial Erythema, edema, and tenderness (stage I); followed within 24 h by paresthesia, marked edema, numbness, and sometimes bullae (stage II); and progressing to gangrene (stage III). Gangrene does not develop unless there is infection. Convalescence may be prolonged for several weeks, months, or even years, during which time there is cold sensitivity, vasomotor instability, hyperemia, and hyperhidrosis (Bashir and Chew 2016). Rest, analgesics, and antibiotics are the mainstays of treatment, which is the same as for frostbite.

During the Korean and Vietnam Wars, thousands of cases occurred, and immersion foot became the major cause of disability. In industries in which workers are required to stand for long periods in cold wet mud or water, as when excavating foundations for new construction, immersion-type injuries may be frequent (Schwartz et al. 1957). Street and sewer workers as well as golf caddies walking for hours on wet grass are also at risk (Chow et al. 1980). For the homeless, immersion foot can be a major health problem (Wrenn 1991), aggravated by lack of

care. Interestingly, immersion foot can also develop in warm water (Humphrey and Ellyson 1997), including the tropics (tropical immersion foot, Table 3).

4.3 Chilblains (Perniosis)

The mildest form of cold injury, chilblains or perniosis, occur as an abnormal reaction to cold in the temperate humid climate of Great Britain and northwestern Europe, where there is a lack of central heating (Heller Page and Shear 1993). Chilblains are less often seen in continental cold climates such as Finland, where well-heated houses and warm clothing are essential. The lesions are reddish blue discolorations that become swollen and boggy, with tense bullae and later ulcerations that may result in scarring. Often, chilblains are superimposed on a background of acrocyanosis or erythrocyanosis. Lesions occur especially on the dorsa of the proximal phalanges of the fingers and toes, heels, lower legs, thighs, nose, and ears. The shiny red plaques itch and burn severely. Chilblains are particularly frequent in children, where they tend to start at the beginning of winter. In adults who work outdoors, chilblains often seem to start in the spring months. Genetic factors are often apparent, e.g., acrocyanosis as an underlying factor. Differential diagnoses of idiopathic pernionsis include chilblain lupus erythematosus (Millard and Rowell 1978), sarcoidosis, and several other entities which have to be considered clinically, histologically, and serologically (Crowson and Magro 1997). Moreover, some patients with chilblains suffer from associated (nonlupus) connective tissue disease or hematologic malignancies (Cappel and Wetter 2014).

The most important point in management is prophylaxis with warm housing, warm clothing, and regular exercise. Once chilblain has appeared, treatment is mainly symptomatic with local application of an antipruritic. Nifedipine may be effective in the treatment of severe recurrent perniosis (Dowd et al. 1986). In seven of ten patients, clearing ranged from 8 days for lesions of the hands to 23 days for foot lesions (Dowd et al. 1986).

4.4 Pulling Boat Hands

This dermatosis has been described from coastal New England (Toback et al. 1985). Erythematous macules and plaques developed on the dorsa of the hands and fingers of instructors and students after 3–14 days aboard a pulling boat. Later, small vesicles appeared, accompanied by itching, burning, and tenderness. Subjects had been exposed to high humidity, cool air, and wind. This was considered an ideal setting for the development of this nonfreezing type dermatosis. Hours of vigorous rowing provided additional repetitive trauma. Some of the patients also had a history of frostbite and Raynaud's phenomenon. This syndrome may be caused by a combination of nonfreezing cold and the mechanical effects of rowing (Toback et al. 1985).

4.5 Other Reactions to Cold

Cold urticaria and Raynaud's phenomenon are dealt with later in this book. The reader interested in other types of abnormal reactions to cold (Table 3) is referred to major textbooks of dermatology, e.g., the article of Bashir and Chew (2016), or a recent systematic review on freezing and nonfreezing cold weather injuries (Heil et al. 2016).

5 Other Atmospheric Factors

A low water content of ambient air may cause, or contribute to, occupational dermatoses, as emphasized by Rycroft and coworkers (Rycroft and Smith 1980; White and Rycroft 1982; Rycroft 1984, 1985), see below. In addition, low temperature, wind, electromagnetic fields, or electrostatic charge have been claimed to be ambient factors important to some skin conditions.

5.1 Low Outdoor Humidity

The following two measures are most commonly used to quantify the humidity of air: absolute humidity (AH) is the mass of water vapor present in a unit volume of the atmosphere and is mostly

measured as mg/l. Relative humidity (RH) is the ratio of the quantity of water vapor present in the atmosphere to the quantity which would saturate it at the existing temperature (expressed as %). Thus, with a given quantity of water per air volume (AH), RH depends on the temperature: the higher the temperature, the greater the water-holding capacity of the atmosphere and the lower the RH, and vice versa.

Low humidity of the air is believed to cause dehydration of the horny layer and impairment of the epidermal barrier function (Agner and Serup 1989), i.e., increased irritability of the skin. Subclinical xerotic changes may occur within hours of exposure and are more pronounced in atopics (Eberlein-König et al. 1996).

Persons working and living in (sub-) polar or even temperate climates with a strong continental influence (prolonged periods of dry, frosty weather in winter) are regularly exposed to low atmospheric humidity, such as 10 mg/l or less. Clinical experience from these countries gives a strong hint on the contributing role to irritant hand dermatitis of these meteorological condition (Kavli and Førde 1984), which has recently been confirmed by an epidemiological study (Uter et al. 1998) and is in good accordance with some experimental data (partially reviewed by Uter et al. (1998)).

Interestingly, unexposed skin displays an impaired epidermal barrier function also (Agner and Serup 1989), which may indicate that even the wearing of protective (warm, wind-tight) gloves may not completely be able to abolish the effect of low humidity on the skin of the hands. The hands may additionally be exposed to a variety of occupational irritants or to wet work. Thus, as (sub-) clinical irritation is known to often be a multifactorial process (Malten 1981), this fairly inalterable environmental condition puts extra emphasis on the necessity of adequate skin protection, be it domestic or occupational.

5.2 Other Ambient Factors

Several authors of experimental studies have attributed negative effects on epidermal properties to low temperature. However, absolute humidity is always also low with low temperature, according to the functional relationship mentioned above, and absolute humidity has not been considered in most of these older studies. At present, the effect of temperature concerning irritant damage can most likely be regarded as indirect.

Wind speed had been found to be a significant risk factor of the occurrence of "dry flaking" facial skin (Cooper et al. 1992). Parish, who exposed the hands of volunteers to "a cold dry wind" 3 h daily, found visible alteration (roughness, desquamation) and impaired lipid and enzyme composition of the epidermis after a few days (Parish 1992).

In addition to natural environmental conditions, anthropogenic factors may lead to occupational (skin) disease:

- During work on a television mast, exposure to high levels of ultrahigh frequency radiofrequency radiation (UHF, 785 MHz mean frequency) caused an immediate sensation of intense heating and later transient Erythema, malaise, numbness, and pain (Schilling 1997).
- Similarly, microwave radiation (1–30 GHz) has been reported as a cause of more or less severe burns, lesser injury being followed by paraesthesia (Kennedy 1992).

5.3 Low Indoor Humidity

Below a water content of 10%, the stratum corneum loses its softness and pliability (Blank 1952). The water content of the horny layer remains below 10% when the relative humidity is less than 50% at room temperature (Rycroft 1985). High temperature and air flow accentuate the drying of the horny layer. An open-plan office next to, or under, the ventilation system, where warm, un-humidified air is introduced into the room is a typical working site where the risk of low humidity dermatosis is apparent. Low humidity dermatoses can be accentuated by small irritant or hygroscopic airborne particles, such as textile particles, dust from ceramics, small particles from paper cutting, and fine angular, hygroscopic particles, as in a soft lens factory

(Rycroft 1984, 1985). Domestic and general climatic conditions can potentiate low humidity occupational dermatoses: Air-conditioned buildings have a low relative (and absolute) humidity, and in temperate areas such as Scandinavia, the low humidity/high temperature indoor environment is accompanied by low humidity/low temperature outdoor climate during the winters, which also has a drying effect on the stratum corneum (see above). The empirical threshold value of 40% relative humidity reported by White and Rycroft (1982) corresponds at a temperature of around 20 °C well with a threshold value of 9–10 mg/l absolute humidity as significant outdoor risk factor (Uter et al. 1998). Some of the occupations where low humidity dermatoses have been reported are listed in Table 6.

These dermatoses are far more distressing than their comparative paucity of physical signs might suggest (Rycroft 1985). Pruritus and burning can be the only sign of low humidity. Puffiness of the cheeks and eyelids has been observed. The skin lesions evolve through dryness of the skin to Erythema and round or oval patches of eczema. Erythema has been accompanied by urticarial whealing possibly secondary to scratching pruritic skin. In some cases, areas covered by clothing have been predominantly involved, while facial itching with diffuse superficial scaling on the cheeks, forehead, and neck have been the main anatomical areas in other instances. Patchy Erythema on the shaved face of male employees has been observed. Fair skinned individuals are at higher risk. Both atopics and nonatopics have been affected.

Differential diagnosis includes a wide variety of possibilities that have to be considered such as inhalable and ingestible allergens, irritant or allergic airborne contact dermatitis, psychological causes, menopausal hot flashes, rosacea and seborrheic eczema, or scabies.

Treatment should include routine use of emollients and increasing the relative humidity indoors to about 50% during the whole low humidity (winter) season.

Table 6 Occupations reported to be related to low ambient humidity

Type of work
Office work
Soft contact lens manufacturing
Silicon chip manufacturing
Cabin crew of long distance airplanes
Resident staff in hospitals and hotels
Travelling salesmen (from automobile heaters)

5.4 Visual Display Units

In some countries, patients often complain of skin symptoms from work with visual display units (VDU; Lidén and Wahlberg 1985; Berg 1989; Eriksson and Stenberg 2006). A study from Sweden among 353 routine office workers showed an increased tendency for seborrheic eczema and nonspecific Erythema. Organizational conditions during VDU work, such as high work load, and inability to take rest breaks, were found to be associated with the reported skin symptoms. A low relative humidity was associated with a diagnosis of seborrheic eczema. However, no associations were found between current field levels of electric or magnetic field and skin diseases/signs or reported symptoms (Bergqvist and Wahlberg 1994). Accordingly, a reduction of electric fields was only weakly associated with an improvement of symptoms attributed to VDU-work (Oftedal et al. 1995). Possibly independent from the initial cause of skin problems, psychological conditioning may lead to a perpetuation of symptoms even without exposure, as illustrated by experiments with affected office workers (Swanbeck and Bleeker 1989). In some cases, perceived symptoms, attributed to VDU-work or other indoor factors, may be part of the ill-defined "sick building syndrome" (Stenberg et al. 1994; Norbäck 2009).

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