Skin injury due to mechanical and thermal trauma is very common. In mechanical injuries, hyperpigmentation or hyperkeratosis, or a combination of both can occur. Corns and calluses are not only sometimes painful, but also can cause significant morbidities and even mortalities in immunocompromised patients such as diabetics. Erythema ab igne is a facultative precancerous dermatosis.

Burns can cause severe damage to all tissue layers. Rapid early treatment can save life and preserve proper functioning. Children are prone to severe systemic damage even after burns affecting small areas. If 5% or more of the body surface is burned, hospitalization is required.

Cold injuries are common. A cold injury is a combination of direct cryogenic cellular effects and ischemia. Injuries range from mild frostbite to more severe and chronic chilblains and cold-induced panniculitis, and severe congelation. In any patient with cold injury, early but mild rewarming is urgently needed.

Mechanical damage to the skin
Mechanical factors act morphogenetically. To a certain extent the skin may adapt to mechanical stresses, reacting with various reaction patterns ranging from hyperpigmentation to ulcerations and hyperplasia. The type of skin reaction depends on the amplitude and frequency of the factor acting on it as well as on the direction of force (Kligman et al. 1985).

The following presents a number of skin disorders that are caused by mechanical factors. A more detailed description of these is not within the scope of this article, however. We have therefore chosen not to include skin disorders that are aggravated by mechanical factors or urticarial disease and decubital ulcers.
Local hyperpigmentation

**Definition**
Circumscribed hyperpigmentations are macular skin changes that have an atypically darker color than the surrounding skin.

**Etiology and pathogenesis**
Local hyperpigmentation may be caused by chafing or rubbing of clothing or prostheses. Risk factors include having dark skin (skin type ≥ 3 after Fitzpatrick) and obesity (Wollina et al. 2006). Hyperpigmentation is occasionally also caused by persistent chafing in neurodermatitis. There is stimulation of melanin synthesis and pigmentary incontinence.

**Clinical presentation**
Circumscribed gray-black or brown hyperpigmentation, usually with poorly-demarcated borders (Fig. 1), occurring at sites where there is rubbing or chafing.

![Figure 1: Friction hypermelanosis caused by the wearing of clothing; obesity as a risk factor](https://example.com/figure1.jpg)

**Therapy**
Affected areas can gradually become lighter again with careful avoidance of rubbing and chafing. The skin usually does not return to its normal color, however. In tropical countries, the use of bleaching agents on the skin sometimes leads to toxic side effects or contact allergy.

**Differential diagnosis**
Other circumscribed hyperpigmentation disorders such as melasma, post-inflammatory hyperpigmentation after toxic skin damage, or adverse drug reactions should also be considered.

Black heel (synonyms: pseudochromidrosis plantaris, hyperkeratosis haemorrhagica)

**Definition**
This is a circumscribed, blackish hyperpigmentation on the heel of the foot.

**Etiology and pathogenesis**
There is bleeding into the horny layer from sudden strong mechanical stress and rupture of small vessels (e.g., in sports involving jumping or sudden stopping on the heel or toes).

**Clinical presentation**
Black heel is common in runners and ball players. The disorder presents with bluish-black, usually painless maculae or striations on the calloused heels or toe pads (Adams 2002; Karamfilov and Elsner 2002). Splinter hemorrhages of the nail plate may be seen. Histological examination reveals deposits of coagulated blood with a positive iron reaction in the horny layer (Kligman et al. 1985). Benzidine stain can be used to detect hemoglobin deposits (Hafner et al. 1995).

**Differential diagnosis**
Melanocytic changes, tattoos, and parasites (tungiasis) should be considered in the differential diagnosis. Dermatoscopy with oil immersion can be useful.

**Therapy**
The disorder does not require treatment.

Blisters due to mechanical causes

**Definition**
Blisters are fluid-filled sac-like lesions that can occur at various sites on the skin.

**Etiology and pathogenesis**
Blisters are caused by shearing forces, heat, or rubbing under pressure, and usually occur on the feet. Typical situations in which blisters form include walking for longer periods of time and wearing ill-fitting shoes. Using the hands for certain tasks (shoveling, hoeing) and leisure activities (tennis, rowing) can also cause blisters. Blisters form in the subepidermis.

**Clinical presentation**
Solitary, taut and painful blisters at contact sites.

**Therapy**
Larger, taut blisters can be punctured under sterile conditions or opened laterally. The roof of the blister should be preserved. If there is a risk of infection, a disinfectant solution such as PVP iodine or polyhexanide should be used and the blister covered with a dressing (Kligman et al. 1985; Adams 2002; Karamfilov and Elsner 2002).
**Differential diagnosis**

Other disorders that should be taken into consideration include bullous skin diseases, bullosa diabetorum, and bullous fixed drug eruption. A bullous ictus reaction is also possible. Bullous toxic contact dermatitis may be distinguished given a more mild inflammation of the surrounding area.

**Callus (synonym: callosity, callositas)**

**Definition**

A callus is an area of hardened skin that is mainly caused by rubbing.

**Etiology and pathogenesis**

A callus can occur with continuous mild or moderate rubbing as a reactive epidermal hyperplasia with hyperkeratosis at sites under mechanical stress (see Fig. 2). Histology shows massive orthohyperkeratosis with variously severe acanthosis.

**Clinical presentation**

Calluses have a predilection for palmoplantar surfaces. Bone deformities such as hallux valgus contribute to their formation. Hyperkeratosis and hyperpigmentation can occur in combination.

Certain occupations are more commonly associated with the development of calluses: guitarists with calluses on the fingertips, chain-smokers with calluses on the thumb (“cigarette lighter’s thumb”). Of historical interest are pretibial calluses, sometimes with mechanical hypertrichosis caused by inflammation. Prepatellar, gray-yellow hyperkeratosis is also observed in nuns and glabellar hyperkeratosis in worshippers (Kahana et al. 1984; Verma and Wollina 2008). Joggers and basketball players can develop hyperkeratotic patches on the medial aspects of the great toes; there is also a tendency in patients with rheumatoid arthritis. Calluses on the finger joints are seen in bulimia (Russell’s sign). Insertion of the finger in the mouth to induce vomiting causes calluses due to repeated contact with the teeth (Russell 1979).

Most important are calluses seen on the plantar surfaces in diabetic patients with peripheral neuropathy (Fig. 2). It has been estimated that a callus, with 10 000 steps per day, can be responsible for an added daily stress of up to 18 600 kg (Pataky et al. 2002). Proper removal of calluses on a regular basis can thus protect diabetic patients from developing diabetic foot syndrome (Pitei et al. 1999; Pataky et al. 2002).

**Figure 2: Central ulcerated plantar callus in peripheral diabetic motor and sensory neuropathy**

**Therapy**

Calluses can be removed after soaking the feet in hot water or with a pumice stone. Professional curettage or abrasion are also options. It is important that patients wear shoes that fit properly. Malposition of the feet should be corrected if possible. Surgical treatment of a callus is only appropriate insofar as it is performed to correct malposition (Pitei et al. 1999; Abouaesha et al. 2001).

**Differential diagnosis**

Circumscribed palmoplantar keratoses should be excluded on the basis of patient history and/or histology.

**Clavus (synonym: corn)**

**Definition**

Clavus develops as a circumscribed, sharply-bordered area of hyperkeratosis overlying the bone at pressure points.

**Etiology and pathogenesis**

Clavi arise as roundish, sharply-bordered areas of hyperkeratosis. Persistent pressure causes a central hyperkeratotic plug to develop over the hard underlying surface of a bony projection. Common sites are the toe joints or the lateral surfaces of the toes in the interdigital space.
Clinical presentation
Clavi typically measure 5–8 mm in size, presenting as yellowish, keratotic plaques with an inflammatory edematous base, and containing a funnel-like plug growing downward which is painful to pressure (Fig. 3). Painful fistulous tracts can develop. Clavi are common in women as well as in patients with rheumatism or diabetes. They are caused by ill-fitting shoes, sometimes in combination with sensory neuropathy. Clavi are also ports of entry for severe foot infection in diabetics. Histology reveals plug-like hyperkeratosis of the acanthotic epidermis. In the sub-epidermis there are degenerative connective tissue changes with edema, liquefication, and fibrosing inflammation (Day et al. 2001).

Figure 3: Clavus of the fifth toe on the lateral aspect and a callus on the lateral aspect of the ankle. The different macromorphologies of the two types of lesions can be clearly seen.

Therapy
Topical therapy is unsatisfactory. Salicylic bandages increase the risk of foot infection, especially in diabetic patients. The most important aspect of treatment is to consistently avoid pressure on the feet (felt corn rings). An experienced podologist can also help. Patients should be advised to wear properly fitting shoes. Surgical correction, performed by an orthopedic surgeon, may be advisable to correct malposition of the toes and avoid recurrence.

Differential diagnosis
In the commonly occurring planter’s warts, there are characteristic thrombosed capillary loops in the form of brown points and bluish-black striated deposits in the center.

Acanthoma fissuratum
Definition
Fissuring inflammations with marginal hyperkeratosis on areas of the skin in contact with eyeglass frames.

Etiology and pathogenesis
Continued irritation of the skin by a heavy or sharp eyeglass frame can cause painful granulation and callus tissue.

Clinical presentation
Acanthoma fissuratum can occur in people who wear glasses, often behind the auricle and sometimes on the bridge of the nose. There is usually a coffee-bean-like, skin-colored or reddened exophytic nodule on one side with a central indentation or fissure, occasionally with superinfection. On palpation there is pain to pressure. Histology of acanthoma fissuratum shows a granulomatous or fibrosing inflammation with pseudoepitheliomatous epidermal hyperplasia and central epithelial invagination (Cerroni et al. 1998).

Therapy
The best result is achieved by adjusting the frame to relieve pressure. Topical therapy with a steroid may also be attempted. Excision, if necessary, may be performed during the inflammation-free interval.

Differential diagnosis
Vegetating pyoderma, flat basal cell carcinoma, psoriasis, eczema, cutaneous lymphoma.

Chewing pads, knuckle pads
Definition
Calluses near joints, usually on the fingers.

Etiology and pathogenesis
Epidermal hyperplasia is caused by repeated and persistent rubbing, chewing or massaging of the affected skin areas which are a sign of a tic, a psychological disorder, or disease. Knuckle pads have also been described as a “side effect” of playing video games (Rushing et al. 2006).

Clinical presentation
Knuckle pads are rare, circumscribed, asymptomatic hyperkeratotic areas occurring on the extensor surfaces of the fingers or sometimes on the dorsal aspects of the hands. They usually have a slight brownish hyperpigmentation. The patient is not always aware of any connection with tic-like behaviors. Depending on the mechanical cause, epidermal or reactive dermal calluses (especially with chewing or biting of the skin) can occur. There is always an external cause – unlike in benign fibromatosis of the fingers (see below). If the mechanical cause is not eliminated, the disorder becomes chronic (Meigel and Plewig 1976; Wollina and Rülke 1994; Wollina 2005).

Therapy
Psychological counseling, if necessary with psychiatric treatment, may be needed. For local therapy, covering the site with a dressing may be useful in stopping mechanical irritation and thus aid healing.
**Differential diagnosis**
Pachydermodactyly, sarcoidosis, polyarthritis, juvenile hyaline fibromatosis (Murray's syndrome).

**Pachydermodactyly (synonyms: digital fibromatosis, pseudo-knuckle pads)**

**Definition**
Pachydermodactyly is a symmetrical acral, painless, fibromatous, and progressive tumor involving the development of connective tissue plaques and diffuse swelling.

**Etiology and pathogenesis**
The etiopathogenesis is largely unknown. This tumor usually occurs sporadically, more often among men, and only rarely with familial incidence. There is no association with mechanical factors (Al Hammadi and Hakim 2007).

**Clinical presentation**
Clinical examination reveals firm intradermal plaques. The plaques are skin-colored, not painful to pressure, and are more pronounced on the finger joints. Histology shows densely arranged, aggregated and interwoven bundles of thickened collagen fibers. The fibrosis involves the sweat glands and can even extend into the subcutis (Kopera et al. 1995; Tompkins et al. 1998).

The disorder usually manifests during the second decade of life. It may be associated with primary hypertrophic osteoarthropathy or arthritis. It is also sometimes associated with seborrhea, cutis verticis gyrata, focal hyperhidrosis, and drumstick fingers (Al Hammadi and Hakim 2007).

**Differential diagnosis**
Knuckle pads, rheumatoid arthritis, Murray's syndrome, pachydermoperiostosis.

**Therapy**
There is no known effective causal therapy.

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**Lipoatrophia semicircularis**

**Definition**
This is a characteristic atrophy of the subcutaneous adipose tissue on the anterior aspects of the thighs.

**Etiology and pathogenesis**
The predominant view is that the disorder arises from mechanical causes, i.e., atrophy of fatty tissue results from repeated microtrauma. Disrupted microcirculation and pressure play a role – e.g., from the edge of a desk if the patient is seated too high or by the wearing of very tight clothing (Nagore et al. 1998; Gruber and Fuller 2001; Herane et al. 2007). Another possibility under discussion is lipolysis occurring from computer-generated electromagnetic radiation (Quatressoz and Piérard 2006).

**Clinical presentation**
A semicircular, band-like depression is found on the skin on the anterior aspect of the thigh. Clinical signs of inflammation are absent. The disorder is especially common in people who work in offices (Hermans et al. 1999). Histology shows subcutaneous fatty tissue atrophy (Nagore et al. 1998).

**Therapy**
There is no known treatment for the cause of this disorder. Symptoms may be treated by injecting fillers or autologous fat.

**Differential diagnosis**
Local lipoatrophy due to insulin or glatiramer injection (Wollina 2006).

**Subcutaneous emphysema due to physical causes**

**Definition**
Subcutaneous edema caused by gas insufflation.

**Etiology and pathogenesis**
This is a very rare, spontaneously occurring condition. It usually occurs after diagnostic or therapeutic procedures (catheter), fractures (orbital fractures) or other injuries (e.g., dental procedures). In physical variants it is assumed that a valve mechanism develops from microtrauma which contributes to air insufflation. An association with pneumomediastinum or pneumothorax is most common (Beck et al. 2002; Verma 2007).

**Clinical presentation**
In physical, non-infectious forms of subcutaneous emphysema, the overall condition of the patient is less severely affected than after a gas burn, for instance. Nevertheless, the swelling can take on monstrous proportions and can be potentially alarming for the patient. Subcutaneous swelling is seen, but erythema is absent. Palpation can elicit a slight crackling noise from the rising gas (Verma 2007).

**Therapy**
Treatment of the underlying disease and elimination of potential mechanical causes (catheter placement) have priority. Subcutaneous catheterization to release air is an option (Beck et al. 2002).
Differential diagnosis
Gas burn, edema, superior vena cava syndrome.

Skin disorders due to thermal causes
The skin is a central organ in the regulation of warmth. Heat entering through the skin can only be partially compensated by sweating and increased circulation. The same is true for cold entering the body via contact or convection. The human body can tolerate only a narrow range of temperature (Kligman and Kligman 1984).

Heat-related skin injury

Erythema ab igne (synonyms: erythema e calore, Buschke’s heat melanosis)

Definition
Erythema ab igne is a form of heat melanosis caused by repeated exposure of the skin to heat.

Etiology and pathogenesis
Repeated, prolonged heating of the skin causes vascular dilation and hyperpigmentation of the epidermis. Typically there is persistent reddish-brownish, reticulated erythema at contact sites, clearly distinguishing this form of hyperpigmentation clinically (Fig. 4) from mechanical forms (see above).

Figure 4: Erythema ab igne with typical reticulated hyperpigmentation

One classic example of erythema ab igne occurring in Tibetan monks presents on the abdomen from the use of a heating stove. In Europe and the United States, the disease is somewhat more common in geriatric patients, related to the use of heating blankets or pillows.

Leaning against a tiled stove or heater can also induce erythema ab igne (Raza et al. 2007; Wollina et al. 2007). Erythema ab igne has also been reported after contact with personal computers (Maalouf et al. 2006) and following pacemaker implantation (Gensch and Schmitt 1981), although these occurrences are rare.

Clinical presentation
One typical sign of the disorder is reticulated hyperpigmentation which is initially reddish and later brownish. The changes are asymptomatic. Hyperpigmentations can occur at various sites (abdomen, lower legs, or back) depending on the heating source. Carcinoma in erythema ab igne has also been reported after a latency period of decades (Peterkin 1955; Wollina et al. 2007).

Therapy
At present there is no known effective therapy. Periodic follow-up is recommended to avoid missing a tumor.

Combustio and ambustio (synonyms: burn and scald injuries)

Definition
Thermal damage to various skin layers and to the entire organism related to the extent and severity of thermal tissue damage.

Etiology and pathogenesis
Causes include direct exposure to a flame, a gas explosion, hot metal, liquid, or steam, UV radiation, tar/asphalt, or an electric current. There are also reports of burn injuries from magnetic resonance imaging. Resonant conductive loops form, e.g., from ECG electrodes, which can produce a burn injury (Dempsey et al. 2001). Thermal trauma to cells occurs at temperatures of 65 °C and higher. Coagulation necrosis arises from denaturation of proteins. The destruction of capillaries triggers an inflammatory cascade leading to a loss of plasma and electrolytes in the interstitial space, leading in severe cases to hypovolemic shock. It is important to note that the initial burn injury can worsen over the next 72 hours. A scald can occur after only three seconds of contact with fluids at temperatures of 60 °C or more.

Clinical presentation
Burns and scalds are common, careless injuries, often occurring at home or during free-time. Burn and scald injuries are very common during early childhood. They can also occur at the workplace or in war. In Germany, the estimated annual incidence is about 2/100 000 in the population (Germann 2004).
Burns/scalds are divided into three grades (Tab. 1) by the depth and intensity of tissue damage. The extent is estimated by Wallace’s “rule of nines” (Tab. 2).

Table 1: Pathogenesis and clinical symptoms by degree of severity of thermal injury

<table>
<thead>
<tr>
<th>Degree of Burn</th>
<th>Pathogenesis and Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>First-degree burn – erythema</td>
<td>Painful reddening of the skin without blistering is suggestive of a first degree burn (Fig. 5). Erythema results, after a delay, from hypervolemia.</td>
</tr>
</tbody>
</table>
| Second-degree burn – blistering | Second-degree burns are divided into grades a and b:  
  - Grade 2a, superficial dermal burn with blistering. Rupture of the blister can make the burn surface moist and glistening and involves a risk of infection. Hair cannot be epilated without pain.  
  - Grade 2b, deep dermal burn, has a white and moist wound base underneath the blisters. There is hair loss, especially of vellus hair. Destruction of nerve endings reduces the sensitivity of the skin to touch. Grade 1 and 2a burns typically heal without scarring, while grade 2b may heal without scarring. Pigmentary changes can develop. |
| Third-degree burn – necrosis | Total dermal burn. The injury destroys all of the skin adnexa. Denaturation of proteins creates a dry, white wound base with a thicker consistency. There is no sensitivity to touch. Re-epithelization does not occur spontaneously (Fig. 6). |
| Fourth-degree burn – charring | Fourth-degree burns cause charring of all layers of the skin and deeper lying tissues (muscles and bone). The burn progressively extends deeper. Determine degree, site and extent of burn, and need for immediate hospitalization. |

Figure 5: First-degree burn after incorrect use of intense pulsed light (IPL) therapy in a cosmetic studio

Figure 6: Third-degree burn after two weeks; necrosis with demarcation. Spontaneous healing does not occur in such injuries. Extensive necrectomy is followed by a mesh graft transplant.

Table 2: Rule of nines for estimating affected body surface area (in %)

<table>
<thead>
<tr>
<th>Body part</th>
<th>Newborn</th>
<th>Infant 1 year</th>
<th>Child 5 years</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head</td>
<td>21</td>
<td>18</td>
<td>14</td>
<td>9</td>
</tr>
<tr>
<td>Trunk, ventral</td>
<td>16</td>
<td>18</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td>Trunk, dorsal</td>
<td>16</td>
<td>18</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td>Arm</td>
<td>9.5</td>
<td>9</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Leg</td>
<td>14</td>
<td>14</td>
<td>16</td>
<td>18</td>
</tr>
<tr>
<td>Genitals</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>
Symptoms of a burn injury may be summarized as a “burn disease.” The acute shock phase begins within the first 48 hours (wound shock); this is followed by the latent (catabolic) shock phase in the first two to four weeks after the trauma until healing. During this time there is a risk of secondary shock related to the extent and degree of the burn. The risk is high in children with burns covering 10 % or more of the body surface area, and in adults with burns on 20 % or more of the body. The mechanism is hypovolemic shock with release of vasoconstrictive mediators that can also trigger edema at sites away from the burn. Loss of fluid is at its maximum immediately after the injury and about two days afterward. Metabolic acidosis, hypoxia, and microthrombosis jeopardize the function of the shock organs - the kidneys, lungs, and liver. The repair phase can overlap with the latency period and is not time-limited. Secondary infections can produce signs of septic shock.

Hyperthermia, pallor, cold acral regions, normotonic tachycardia, thirstiness, and restlessness are signs of impeding shock. Shock itself is marked by sweating, hypotonic tachycardia, and cold acral regions. Hiccups, seizures, and vomiting are associated with an unfavorable prognosis. Especially during the first 48 hours there can be oligemia and impending insufficient supply to the brain, kidneys, liver, muscle and gastrointestinal tract. Acute anuria can occur as an expression of kidney failure. Affected patients can die of uremia (Kibbi and Tannopus 1998; Sheridan 2001).

Even today, wound infection is a dreaded complication of any burn injury. Bacterial colonization of the burn site can occur with Gram-positive organisms such as staphylococcal bacteria, Gram-negative organisms, or saprophytic fungi such as Candida and Mucorales (Ledgard et al. 2008; Saffle et al. 2008). Bacterial endotoxins and exotoxins can also trigger symptoms of shock that can be associated with purpura fulminans (Hassan et al. 2008). Bacterial colonization leads to delayed wound healing.

Wound diphtheria and scarlet fever can also occur. Dreaded complications include stomach and duodenal ulcers with a tendency to bleed. Bronchopneumonia and bronchitis have also been reported.

The prognosis depends largely on the extent and degree of the burn. Burns that are critical are those in which more than > 20 % of the body surface area (BSA) in an adult or > 10 % in a child, or > 5 % in a young child, is involved. Burn shock and wound infection, older age, organ and circulatory disease, pregnancy and puerperium are associated with an unfavorable prognosis (Kligman and Kligman 1984; Kibbi and Taurons 1998).

Therapy
An estimated 80–90 % of all burns/scalds can be treated on an outpatient basis. The following early measures are recommended (Tab. 3). Local therapy should be dry, clean, and sterile. In outpatient dermatology, that is often sufficient for minor injuries.

In the clinic, shock therapy must be administered to adults with involvement of 15 % or more of the BSA and 8 % BSA involvement in children. Ringer’s lactate infusion solution is administered based on the Baxter formula: 4 x kg body weight x % burn surface = ml Ringer’s lactate in 24 hrs. (for adults). Adequate pain therapy must be ensured. For second-degree burns or worse, pain therapy should be administered intravenously analogous to the WHO analgesic ladder for cancer pain management.

Table 3: Early treatment of burn/scald injuries (based on the recommendations of the German Medical Society for Burn Injuries 2001)

- Eliminate the heat source or harmful agent, remove hot clothing, turn off electricity, etc., protect self
- Check vital signs
- For minor injuries, cool with tap water until pain subsides; use caution with extensive burns, avoid under-cooling
- Estimate the extent of damage as a percentage of body surface area and depth of damage in degrees.

Initial treatment following a burn injury consists of prompt cooling of the affected part of the body. Prompt cooling with cold water prevents worsening of the burn and alleviates pain. If the patient is on fire, the flames must first be extinguished with a blanket, by rolling, or with a fire extinguisher. Blisters should not be opened given the risk of infection. For minor burns/scalds, carbonate dressings or gauze bandages with silver sulfdiazine or fusidic acid can be applied to erosive surfaces.

Take care not to overestimate the therapy needed by a burn victim!

The following types of patients should be admitted to a burn care unit:
- Any patient with burns on the face/neck, hands, feet, anogenital region, axillae, areas overlying large joints, or other complicated sites
- Adults with 2nd degree burns on 15 % BSA or more
- Adults with 3rd degree burns on 10 % BSA or more
- Patients with accompanying mechanical injuries
- Any patient with respiratory injury
- Patients with pre-existing disease or patients under 8 years or over 60 years of age (German Medical Society for Burn Injuries 2001)
Extra caution must be taken with children and elderly patients. Shock therapy/prevention must be promptly initiated!

Transfer to a specialized center is accomplished by calling the respective center or, in Germany, by calling a centralized service for bed availability (ZBB) in Hamburg (telephone: 040 42851-3998, -3999, fax: 040 42851-4269, e-mail: leitstelle@feuerwehr.hamburg.de).

Follow-up care
In spontaneous healing of deeper (grade 2b and worse) burns and after skin transplants, scar-tissue contractures may develop. The younger the patient, the worse the scarring. To prevent contractures from forming, measures such as splinting, fixation, and intense movement exercises are needed. Long-term compression therapy for a minimum of six months (several years in children) can help prevent hypertrophic scar formation and keloid scarring. Topical anti-scarring therapies should be used to reduce the toughness and vulnerability of the skin (fragrance-free moisturizers, silicone gel). Specific corrective measures for repairing scars should be performed by a specialized plastic surgeon. Chronic, ulcerating burn scars are facultative precancerous lesions.

Cold-related skin injury

Congelatio (synonym: frostbite)

Definition
Frostbite refers to a cold injury that, depending on the intensity and duration of exposure, leads to reversible or irreversible damage.

Etiology and pathogenesis
Exposure to cold leads to direct cryogenic cell damage through formation of ice crystals in the extracellular space or indirect damage. The latter commonly occurs via vaso-constriction in the arterioles and venules as ischemic tissue damage. Unlike burn injuries, there is no protein coagulation.

Exposure to cold ultimately leads to release of histamine-like substances, prostaglandin, and thromboxanes, leading to vasodilatation, increased capillary permeability with extravasation, inflammation, slowed blood flow, oxygen deficiency, and necrosis. Hence there is a cumulative effect of exposure to cold and ischemia (Murphy et al. 2000).

Clinical presentation
Frostbite depends on the intensity of the cold, duration of exposure, humidity and protective factors such as clothing and movement. The first sign of under-cooling is the appearance of goosebumps (pes anserinus, cutis anserina), resulting from contraction of the Mm. arrectores pili. Cold injuries begin at the terminal vessels in acral regions (frostbite, chilblain). If the entire organism is affected, the patient can freeze to death. The body is then frozen stiff. Contributing factors include acrosphyxia, palmar hyperhidrosis, constrictive or light clothing, physical exertion, exhaustion, alcohol consumption, and blood loss.

A special case is injury caused by landslide or shipwreck, in which suspended animation can occur. Under-cooling is present. At temperatures < 22°C the damage is irreversible. Rectal temperatures < 20°C are a sign of death due to heat loss.

Frostbite injuries may be divided into three grades (Tab. 4).

Generalized symptoms are usually absent, although ventricular fibrillation is a dreaded complication. Subjective delayed sequelae are abnormal sensitivity to cold, metrosensitivity, and dysesthesia. The prognosis is determined by the intensity and extent of cold injury. Secondary infections are a grave complication, as is alcohol consumption, which contributes to generalized under-cooling via vascular dilation and can lead to death (Long et al. 2005).
Table 4: Classification of frostbite by degrees

<table>
<thead>
<tr>
<th>Degree</th>
<th>Description</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>First-degree</td>
<td>Dermatitis congelationis erythematosa</td>
<td>Ischemic constriction of (cutaneous) vessels with whitish discoloration and loss of sensation. Pain in partially frozen tissue. After rapid rewarming, there is temporary severely pruritic or burning erythema. Recovery is complete.</td>
</tr>
<tr>
<td>Second-degree</td>
<td>Dermatitis congelatio bullosa</td>
<td>Rewarming causes subepidermal blistering, sometimes with a serous or hemorrhagic component.</td>
</tr>
<tr>
<td>Third-degree</td>
<td>Dermatitis congelatio escharotica</td>
<td>Occurs with very intense exposure to cold. Necrosis develops. The frostbitten body parts can turn blue or black and become hard and numb. Dry necrosis (mummification) can follow with a blue-black leather-like eschar, or under the influence of bacterial colonization, moist necrosis (gangrene) can develop which is associated with a poorer prognosis. A demarcating inflammation separates necrotic from healthy tissue. Spontaneous separation of devitalized tissue takes a long time (months).</td>
</tr>
</tbody>
</table>

For third-degree frostbite, mummification should be the goal. Wet therapies must be avoided. Early amputation may be considered in patients with moist gangrene. In mummification, amputation should be delayed until the inflammation-free interval, occurring after 1-3 months.

Pernio (synonym: chilblains)

Definition
Chilblains are livid, cushion-like swellings on the skin after cold exposure.

Etiology and pathogenesis
Pernio is autonomic, abnormal vascular functioning that correlates with low temperatures. Especially in the spring and fall, pernio can occur at temperatures just above 0°C. Contributing factors are wearing tight shoes, tight gloves, inadequate clothing, and exposure to damp conditions. Pernio tends to affect adolescents with acrocyanosis, mainly after working outdoors, riding a motorcycle, or after being in cold and humid places. An association with cutis marmorata, erythrocyanosis crurum puellarum, or focal hyperhidrosis is not uncommon. Women are more often affected than men. Genetic factors also appear important. Japanese and African Americans are more frequently affected than white Europeans. Paradoxically, smokers are less susceptible.

Clinical presentation
Chilblains appear primarily on the dorsal aspects of the fingers and toes, on the lower legs, the lateral aspects of the thighs or, in women, on the breasts. Lesions are pillow-like, nodular or papular, blue or red swellings that upon rewarming can be pruritic or associated with burning and pain (Fig. 7). With heat-related hyperemia, the color turns vermillion. Pernio with blistering or secondary ulceration can also occur.

Figure 7: Pernio over the MIP joint of the 3rd and 4th toes as well as over the PIP joint of the great toe

Therapy
To heat the body from the inside, the patient is given warm or hot drinks (tea, coffee). Pentoxifylline and pyridylmethanol can be given to improve peripheral circulation.

For external therapy, rapid rewarming of healthy body parts, while keeping the frozen areas relatively cool, is prudent. Hot baths are contraindicated, as sudden intense rewarming of frozen tissue exacerbates ischemia and resultant tissue damage.

Rewarming can be achieved in moderately warm water (35°C), with a heat lamp (30–40°C), or using warm dressings. Pentoxifylline 40 mg/kg of body weight, aspirin 5 mg/kg of body weight, and vitamin C 50 mg/kg of body weight are recommended (Purkayastha et al. 2002; Long et al. 2005).
Typical histological findings include a combination of dermal edema and a lymphocytic infiltrate in the reticular dermis that is mainly deep and peri-eccrine. The epidermis contains necrotic keratinocytes. Pernio can continue to recur for years (Long et al. 2005).

**Differential diagnosis**

Involvement of the fingers and toes should raise suspicion of chilblain lupus. This is also often associated with lunula erythema, however (Wollina et al. 1999). The symptoms do not entirely disappear during the summer. Spring pernio (papulovesicular lesions mainly on light-exposed areas such as helices, face, neck, dorsal aspects of the hands, forearms, and lower legs) is a form of polymorphous light eruption. On the lower legs, erythema induratum (Bazin’s disease) or sarcoidosis should be considered.

**Therapy**

*Systemic:* increase peripheral circulation with pentoxifylline (30 mg/kg/body weight) or naftidrofuryl (3 x 200 mg/Tag).

*Topical:* the best form of prevention is to avoid cold and damp by wearing appropriate clothing. Treatment consists of warm baths with nicotine acid benzyl ester.

**Pernio follicularis (synonym: cutis anserina perpetua rubra)**

**Definition**

Pernio follicularis involves cold-induced, livid papular perifollicular millimeter-large lesions that contain vertically-standing hairs.

**Etiology and pathogenesis**

The physiological «goosebump reaction» is triggered by a cold stimulus. A persistent reaction apparently occurs with habitual factors such as pyknic habitus. Younger women are more often affected.

**Clinical presentation**

The disorder usually presents on the lower legs and feet, with densely arranged, bright red, pinhead-sized perifollicular papules and vertically-standing hairs due to persistent contraction of the pilar muscles. This can lead to secondary follicle keratosis. Pernio follicularis is a predisposing factor for follicular infections. Symptoms tend to be more mild than in pernio and may even be absent (Kühl and Tronnier 1979; Karamfilov and Elsner 2002).

**Cold panniculitis**

**Definition**

Very rare lobular panniculitis caused by circumscribed cold exposure.

**Etiology and pathogenesis**

In predisposed persons, cold stimulus leads to abnormal microcirculation in the subcutaneous adipose tissue with subsequent panniculitis.

**Clinical presentation**

Cold panniculitis occurs almost exclusively in obese women on the buttocks, lateral thighs, breast, neck, or double chin 48 hours after exposure to the cold. Painful, inflamed indurations occur that resemble erythema nodosum (Page and Shear 1988; Kühl and Tronnier 1979).

**Therapy**

Symptoms may be treated with non-steroidal anti-rheumatic agents. Local ichthyol dressings.

**Differential diagnosis**

Other forms of panniculitis, especially erythema nodosum and atypical borelliosis.

CME Dermatol 2009; 4(1): 4–18

**Keywords**

Burns, cold injury, hyperpigmentation, corns, callosities


References


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CME-Continuing Medical Education

Thermal and mechanical skin injuries

Question 1
Which method can be used in differential diagnosis of black heel?
- a. Dermatoscopy
- b. Bacteriological culture
- c. Congo red stain
- d. Warton-Sterry stain
- e. Sonography (20 MHz)

Question 2
What is the difference between callus and clavus?
- a. There is no difference.
- b. Callus tends to occur over a bony prominence.
- c. Calluses are less common in diabetics.
- d. Calluses are typical signs of abnormal barrier function.
- e. A finger callus is more common in cachexia.

Question 3
Which of the following statements is correct?
- a. Acanthoma fissuratum is a granulating inflammation.
- b. Acanthoma fissuratum is due to mycotic causes.
- c. Acanthoma fissuratum is contagious.
- d. Primary therapy of acanthoma fissuratum is pain therapy.
- e. Acanthoma fissuratum is an occupational disorder in welders.

Question 4
Which of the following applies to knuckle pads?
- a. Knuckle pads are a common geriatric syndrome.
- b. Knuckle pads are caused by collagen thickening.
- c. Knuckle pads are a symptom of video-game playing.
- d. Knuckle pads are associated with arthritis.
- e. Knuckle pads are best treated surgically.

Question 5
Which of the following applies to lipoatrophia semicircularis?
- a. It is most common in drivers.
- b. It is associated with hereditary metabolic disorders.
- c. It is a special form of cellulite.
- d. It is one of the sequelae of traumatic panniculitis.
- e. Asthenic persons are more often affected.

Question 6
A 4-year-old child has a burn on the right arm (grade 1–2). Which of the following statements is true?
- a. This is a minor thermal injury. Hospitalization is not necessary.
- b. Children who have not been immunized against tetanus should be hospitalized.
- c. Hospitalization is only necessary for scalds.
- d. Any small child with involvement of > 5 % BSA must be promptly admitted to the hospital.
- e. First aid consists of cooling with a cooling ointment.

Question 7
You are on call on New Year’s Eve. Around 2 a.m. a young man is admitted with a traumatic tattoo on the face caused by an exploding firecracker. His face also exhibits a moderate redness. You check his immune status. His last tetanus vaccination was about 10 years ago. What immediate measures must be taken? Everything else can wait.
- a. Cool the face with water
- b. Ask to see his chip card
- c. Tetanus booster
- d. Wound smear
- e. Call the surgeon to remove the traumatic tattoo.

Question 8
Erythema ab igne is a chronic thermal skin injury. Which clinical symptom does not apply?
- a. A reticular vessel pattern is found at sites of contact with the heat source.
- b. There is red-brown discoloration of the skin.
- c. Erythema ab igne is usually asymptomatic.
- d. There is unceasing pruritus.
- e. Erythema ab igne is a facultative precancerous lesion.
Question 9
What distinguishes cold injuries from thermal injuries?

a. The skin is more tolerant toward cold.
b. In burn injuries the demarcation of necrosis is detectable later than in frostbite.
c. Protein coagulation does not occur in frostbite injuries.
d. Ischemia plays a more minor role in frostbite injury.
e. Vitamin D therapy is helpful in treating frostbite.

Question 10
Chilblains (pernio) are induced by cold exposure. Which of the following statements is correct?

a. Hypertension can quickly lead to compromised circulation in acral regions.
b. Pernio does not involve vasculitis.
c. Being overweight is protective against cold injuries.
d. Pernio usually affects the face.
e. Alcohol improves circulation in acral regions.