NOTES
BURNS & FROSTBITE

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Damage to skin, underlying structures due to overexposure to harmful conditions
- Burn/frostbite injury → loss of skin function
  - Impaired thermoregulation → loss of body heat
  - Impaired fluid retention → large water, protein losses from skin, affected tissues
  - Loss of microbial barrier function → high risk of infection

SIGNS & SYMPTOMS

- Pain, erythema, blistering, skin layers slough off

DIAGNOSIS

OTHER DIAGNOSTICS

- Clinical presentation
  - Nature of exposure, appearance of wound, depth of damage

TREATMENT

MEDICATIONS

- Analgesia

SURGERY

- Debridement of dead tissue

ACTINIC KERATOSIS

osms.it/actinic_keratosis

PATHOLOGY & CAUSES

- Repeated prolonged sun exposure → small, ill-defined, rough, scaly patches of skin
- Once initial lesions develop, more may follow without additional sun exposure
- UVB radiation → damage to keratinocytes → accumulation of oncogenic changes (e.g. p53 gene mutation) → unchecked proliferation of dysplastic keratinocytes → precancerous lesion

RISK FACTORS

- Fair-skinned individuals; facial distribution, sun-exposed limbs; increased age; immunosuppression; albinism; xeroderma pigmentosum; human papillomavirus (HPV) infection

COMPLICATIONS

- Malignant transformation to squamous cell carcinoma (0.03–20% likelihood)

SIGNS & SYMPTOMS

- Small, rough, scaly skin lesions
- Sandpaper-like sensation felt on palpation
- Induration, tenderness, bleeding → possible malignant transformation

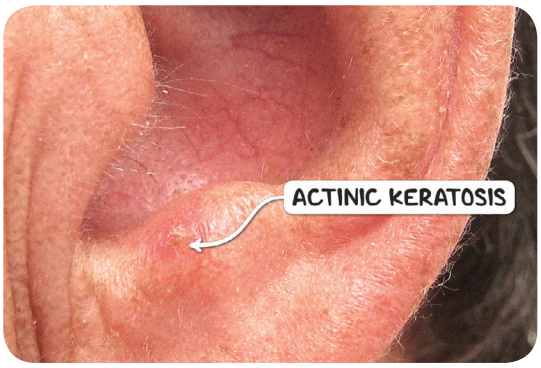

Figure 1.1 The clinical appearance of an actinic keratosis.

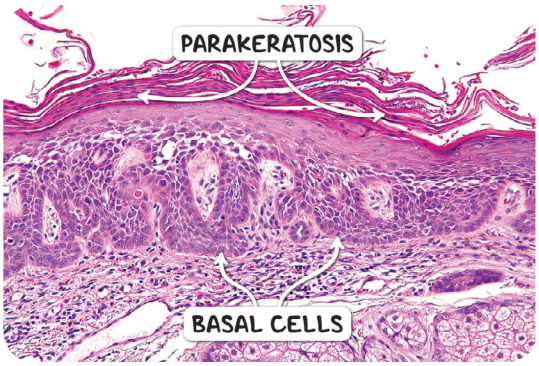

Figure 1.2 The histological appearance of actinic keratosis. There is full thickness epidermal atypia with hyperchromatic basal cells and nuclei in the stratum corneum (parakeratosis).

DIAGNOSIS

LAB RESULTS

- Skin biopsy
  - Exclude malignancy

TREATMENT

MEDICATIONS

- *Topical pharmacotherapy*: 5-Fluorouracil, imiquimod, ingenol mebutate

SURGERY

- Scraping, excision

OTHER INTERVENTIONS

Prevention

- Avoid excessive sun exposure, use sunscreen

Dermatologic

- Cryotherapy (liquid nitrogen), photodynamic therapy, electrodessication

BURNS

osms.it/burns

PATHOLOGY & CAUSES

- Tissue destruction due to exposure
  - Heat, electricity, chemicals, radiation
- Burn injury → loss of skin function
- Impaired thermoregulation → loss of body heat
- Impaired fluid retention → large water, protein losses from skin, affected tissues
- Loss of microbial barrier function → high risk of infection
- Cell survival favoured by moist environment, aseptic conditions, good blood supply

TYPES

Thermal burns

- Contact with heat/heated objects, fluids
- > 44°C/111.2°F
  - Proteins denature, break down → cell damage
- Amount of tissue destruction determined

by temperature, duration → injury diminishes outwards as heat disperses around central site
- *Zone of coagulation (ischemia)*: area of maximal damage; no remaining tissue perfusion → irreversible cell damage → coagulative necrosis
- *Zone of stasis (edematous)*: surrounds coagulation area, microvascular sludging, thrombosis → decreased perfusion → progressive tissue necrosis; cellular death within 24–48 hours without treatment; early intervention may save significant amounts of tissue
- *Zone of hyperemia*: surrounds zone of stasis; inflammation → vasodilation, increased capillary permeability → erythema; tissues still viable → recovery likely

Chemical burns
- Exposure to corrosive substances (e.g. acids, bases, oxidizing/reducing agents, solvents, alkylants, chemical weapons)
- *Severity*
  - Alkali > acid; warmer temperature; greater volume, concentration, contact duration; specific mechanism of chemical action; degree of tissue penetration
- Occur immediately on contact, may continue to progress for some time
- May not be immediately evident
- May diffuse to deeper structures without initial damage to skin surface

Electrical burns
- Passage of electricity through tissue → rapid injury
- Subdermal damage significantly greater than superficial injury
- Extent of injury determined by
  - *Current*: higher current → increased lethality/tissue damage
  - *Voltage*: higher voltage → more damage; higher voltage → dielectric breakdown of skin → lowered resistance, greater current flows
  - *Frequency*: very high frequencies → tissue burning; doesn't penetrate deep enough to affect heart
  - *Duration*: longer duration → more tissue damage
  - *Pathway*: current flowing through heart → lethal
  - *Tissue resistance (pathway, depth dependant)*: nerves < blood vessels < muscle < skin < tendon < fat < bone

Radiation burns
- Excessive exposure to radiation
  - *Ultraviolet (UV) light*: sunlight most common cause of radiation, superficial burns
  - *Ionizing radiation (e.g. radiation therapy, X-rays, radioactive fallout)*: skin effects vary from hair loss at 3Gy to necrosis at 30Gy
  - *Microwave burns*

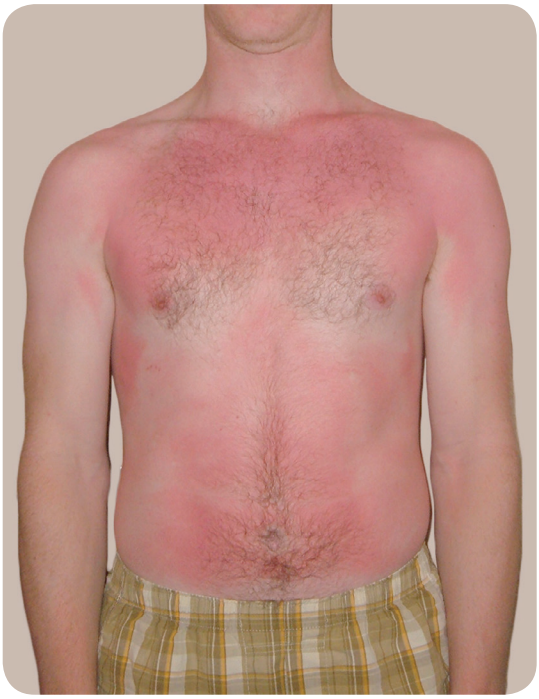

Figure 1.3 An adult male with superficial partial thickness burns to the arms and torso, secondary to sun overexposure.

RISK FACTORS

- Complicated injury
  - Age (< 3, > 60), location (e.g. face, neck, hands, feet, perineum), inhalational injury, associated injuries (e.g. fractures), comorbid disease (e.g. chronic renal failure)

COMPLICATIONS

- Wound contracture/hypertrophic scarring, infection
  - *Most common organisms:* *S. aureus*, *P. aeruginosa*, *C. albicans*
- Systemic effects of severe burns
  - Large burns > 30% of total body surface area → significant inflammatory response → impaired organ perfusion → gastrointestinal (GI) bleeding, renal failure, progressive pulmonary insufficiency
  - Increased levels of catecholamines, cortisol → hypermetabolism. immunosuppression
- Additional injury
  - Singeing of airways → inflammation → eventual compromised airway
  - Carbon monoxide inhalation

BURNS OVERVIEW

	SKIN LAYERS INVOLVED	CLINICAL FEATURES	HEALING
SUPERFICIAL BURN (1ST DEGREE)	Epidermis	Sensation intact → pain, erythematous, blanchable, hair follicles present	5–10 days: no scarring, heals completely
SUPERFICIAL PARTIAL THICKNESS BURN (2ND DEGREE)	Extends into superficial (papillary) dermis	Sensation intact → pain, erythematous, forms blisters containing clear fluid, blanchable, hair follicles present	2–3 weeks: spontaneously re-epithelializes
DEEP-PARTIAL THICKNESS BURN (2ND DEGREE)	Extends into deep (reticular) dermis	Yellow/white, no sensation, minor blanching, blisters, some hair follicles still intact	1–2 months: re-epitheliaizes, hypertrophic scars common, grafting recommended to expedite healing
FULL THICKNESS BURN (3RD DEGREE)	Extends through epidermis, dermis	No sensation, stiff leathery eschar (black/ grey/white/ cherry red in colour), no hair follicles, thrombosed veins visible	Incomplete/months: Re-epithelializes from wound edge, grafting necessary to replace dermal integrity, limit scarring
FULL THICKNESS BURN (4TH DEGREE)	Injury to underlying tissues (fat/muscle/bone)	Black/charred	Requires debridement/ amputation: skin flap for coverage, does not re-epithelialize, cannot graft

DIAGNOSIS

OTHER DIAGNOSTICS

- % of total body surface area (TBSA) affected
  - Doesn't include areas with first degree/superficial burns
  - *Palm size estimation*: size of individual's hand print (palm, fingers) 1% of TBSA
  - *Wallace rule of nines*: each major body part assigned value corresponding to approx. proportion of body surface area

American Burn Association severity classification

- Minor
  - < 2% full thickness burn
  - < 10% TBSA (young/old < 5% TBSA)
- Moderate
  - 2–5% full thickness burn
  - 10–20% TBSA (young/old 5–10% TBSA)
  - high voltage injury, possible inhalation injury, circumferential burn, comorbidities
- Major
  - > 5% full thickness burn
  - > 20% TBSA (young/old > 10% TBSA)
  - high voltage burn, known inhalation injury, significant burns to face/joints/hands/feet, associated injuries

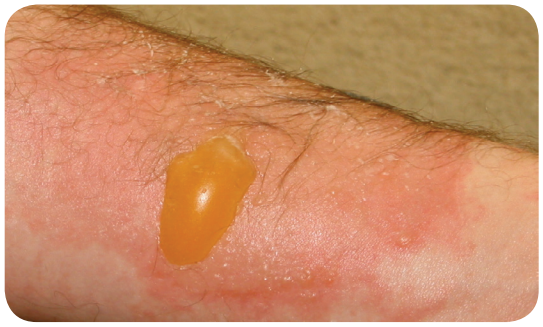

Figure 1.4 A full-thickness superficial burn to the hand.

WALLACE RULE OF NINES

	ESTIMATED BODY SURFACE AREA (%)	
	ADULTS	CHILDREN
LEFT ARM	9	9
RIGHT ARM	9	9
HEAD	9	18
CHEST	9	9
ABDOMEN	9	9
BACK	18	19
LEFT LEG	18	13.5
RIGHT LEG	18	13.5
GROIN	1	1

TREATMENT

OTHER INTERVENTIONS

Intravenous (IV) fluids

- Parkland formula
  - Estimated IV fluid replacement required over initial 24 hours
  - Volume required in 24 hours = 4 x mass (kg) x (% TBSA x 100)
  - Half of requirement given over first eight hours; remainder over following 16 hours

Wound care

- *First degree*: maintain moist skin barrier with antimicrobial burn dressings
- *Second degree*: daily burn dressing change with topical antimicrobial, leave blisters intact unless circulation impaired/overlying joint, inhibiting movement
- *Deep second degree*: prevention of sepsis → antibiotics

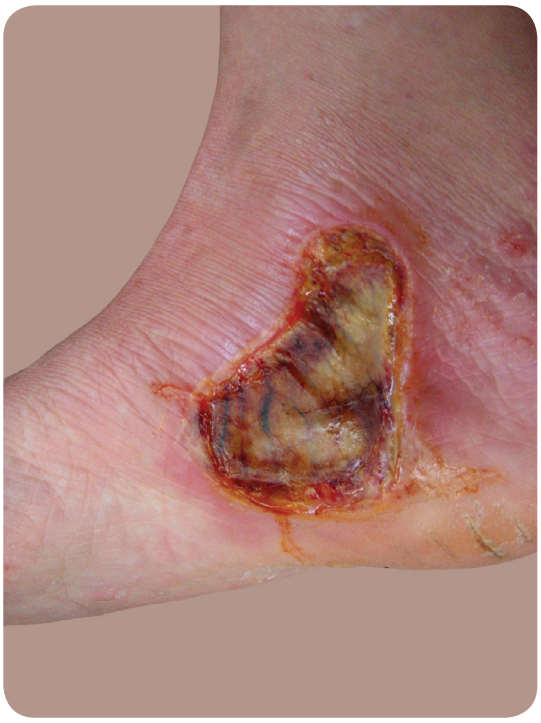

Figure 1.5 A full thickness burn to the medial aspect of the foot.

- Remove dead tissue
  - Surgical debridement, excise to viable (bleeding) tissue

Chemical burn

- Remove contaminated clothing, brush off dry powder
- Irrigate with water for 1–2 hours under low pressure; if elemental metal burn (e.g. sodium, potassium, magnesium, lithium) avoid exothermic reaction with water, soak in mineral oil instead
- Acid
  - Water irrigation, followed by dilute solution of sodium bicarbonate

Electrical burn

- Debride non-viable tissue, repeat every two days
- Monitor for cardiac complications

FROSTBITE

osms.it/frostbite

PATHOLOGY & CAUSES

- Exposure to low temperatures for significant periods of time, subsequent rewarming → tissue damage

Freezing

- Temperatures < −4°C/24.8°F → formation of ice crystals within tissues → damage to cellular membranes, small blood vessels
- Cooling → vasoconstriction, impaired circulation → further cooling, warm blood unable to effectively perfuse freezing extremities

Thawing

- Rewarming → vasodilation → edema
- Poor blood flow through damaged capillaries → ischemia, inflammation, blood coagulation → tissue death
- Thawing → formation of blood clots in small vessels

RISK FACTORS

- Frequently exposed/thermally vulnerable skin (e.g. hands, feet, face); occupational/hobby exposure to low temperature environments (e.g. winter sports enthusiasts, military personnel); circulation-impairing disorders (e.g. Raynaud's phenomenon, diabetes), substance use (e.g. smoking)

COMPLICATIONS

- Hypothermia, compartment syndrome

SIGNS & SYMPTOMS

- Numbness prior to thawing
- White/bluish discolouration of skin
- Swelling/blistering after treatment

DIAGNOSIS

- *Clinical presentation*: physical assessment, classification

DIAGNOSTIC IMAGING

- Technetium (Tc)-99m scintigraphy (SPECT scan)/CT scan
- Assess salvageable tissue; earlier debridement of nonviable soft tissue
- Perfusion/metabolic imaging identifies viable bone, tissue/location autoamputation likely to occur

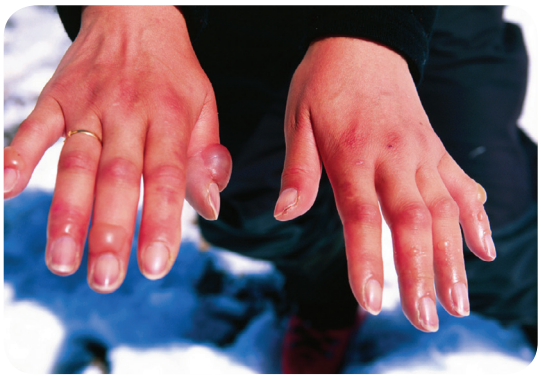

Figure 1.6 The clinical appearance of frostbitten fingers.

CLASSIFICATION OF FROSTBITE

	TISSUE LAYERS INVOLVED	CLINICAL FEATURES	CLINICAL COURSE
FROSTNIP	Superficial skin damage, without ice crystal formation	Pallor, numbness, reverse quickly on rewarming	Rapid recovery
1ST DEGREE	Superficial skin damage	Central pallor, anesthesia with surrounding edema	Skin surface may slough off, damage not permanent → full recovery
2ND DEGREE	Full thickness skin injury	Within 24 hours after exposure: large blisters containing clear fluid form, surrounded by edema, erythema	Blisters may form eschar → sloughs off to reveal healthy granulation tissue → distal tissues/nails may be destroyed
3RD DEGREE	Full thickness skin injury, may affect under tissues	Deeper than 2nd degree injury, smaller blisters form → may hemorrhage; skin forms black eschar over weeks	Long term ulceration, lesions on intermediate body parts → loss of function/autoamputation
4TH DEGREE	Injury to underlying tissues (fat/muscle/bone)	Complete tissue necrosis, painless rewarming, mummification occurs in 4–10 days	Sepsis likely, autoamputation may occur over 1–2 months

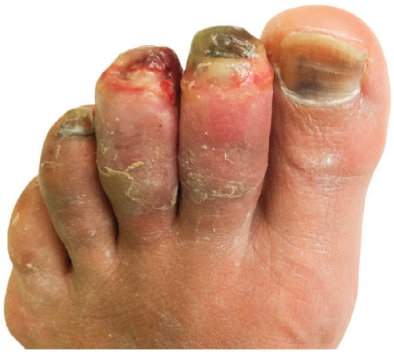

Figure 1.7 Toes three weeks following frost bite.

TREATMENT

MEDICATIONS

Initial thawing

- Analgesia
  - Nonsteroidal anti-inflammatory drugs (NSAIDs)/opioids
- Pharmacological adjuvants (severe cases, grade 2+)
  - *Antithrombotics:* tissue plasminogen activator, heparin for risk of amputation
  - Blood vessel dilator: iloprost
  - Sympatholytic drugs → counteract peripheral vasoconstriction
  - High risk of infection → antibiotic prophylaxis (e.g. penicillin G)

SURGERY

- Debride dead tissue
- *Escharotomy*: release restrictive eschars
- *Fasciotomy*: compartment syndrome

OTHER INTERVENTIONS

General measures

- Do not rewarm if possibility of refreezing exists (worse tissue damage)
- Do not walk on frostbitten feet/rub frostbitten hands (worse tissue damage)
- Avoid using stoves/fires to reheat insensate limbs (avoid thermal damage)

Initial thawing

- *Temperature*: immerse in 37–39°C/98.6–102.2°F agitated water; maintain steady temperature
- *Duration*: 10–30 min with povidone iodine/chlorhexidine antiseptic

SUNBURN

osms.it/sunburn

PATHOLOGY & CAUSES

- Radiation burn of living tissue due to excessive exposure to UV radiation
  - Burning may occur in 15 minutes of sunlight exposure in high UV radiation areas/seconds of non-shielded welding arcs
- UV light radiation overexposure
  - Initial direct DNA damage (formation of thymine dimer) → activates cellular response mechanisms → DNA repair/inflammatory response, cell death via apoptosis
  - Within one hour mast cells degranulate → release of histamine, serotonin, tumor necrosis factor (TNF) → prostaglandin, leukotriene synthesis → neutrophilic, lymphocytic infiltrate → further inflammation
- UV exposure → activation of genes to produce melanin → absorbs UV wavelength light → acts as photoprotectant

RISK FACTORS

- Outdoor work/sports, fair skin, very young/old age, genetic defects in DNA repair, use of photosensitizing medication

COMPLICATIONS

- Increased risk of skin cancers (e.g. melanoma; basal-cell, squamous-cell carcinoma)

SIGNS & SYMPTOMS

- Initial erythema, heat given off by increased blood flow to area due to vasodilation
- Pain proportional to severity of exposure
- Blistering, swelling, edema, peeling skin, fever, chills

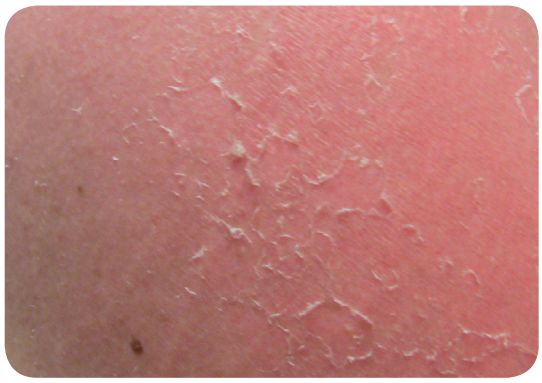

Figure 1.8 Desquamation (peeling) of the skin following sunburn.

DIAGNOSIS

OTHER DIAGNOSTICS

- Clinical presentation (similar to thermal burn)
  - Superficial (first degree) → affects only epidermis (erythematous)
  - Superficial partial thickness (second degree) → affects dermis (forms blisters)

TREATMENT

MEDICATIONS

Analgesia

- hydrocortisone cream, NSAIDs

OTHER INTERVENTIONS

- Protect burnt skin with loose fitting clothing when outside to prevent further damage

Analgesia

- Cool baths/showers, soothing skin moisturizers

Prevention

- Avoid peak UV radiation intervals (10:00 AM to 4:00 PM), wear appropriate clothing (e.g. long-sleeved shirts, long trousers, wide-brimmed hats, sunglasses), broad-spectrum sunscreen on any exposed skin