INTEGUMENTARY SYSTEM

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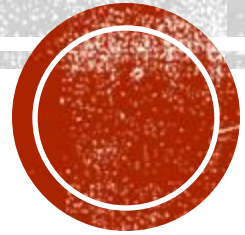


OBJECTIVES

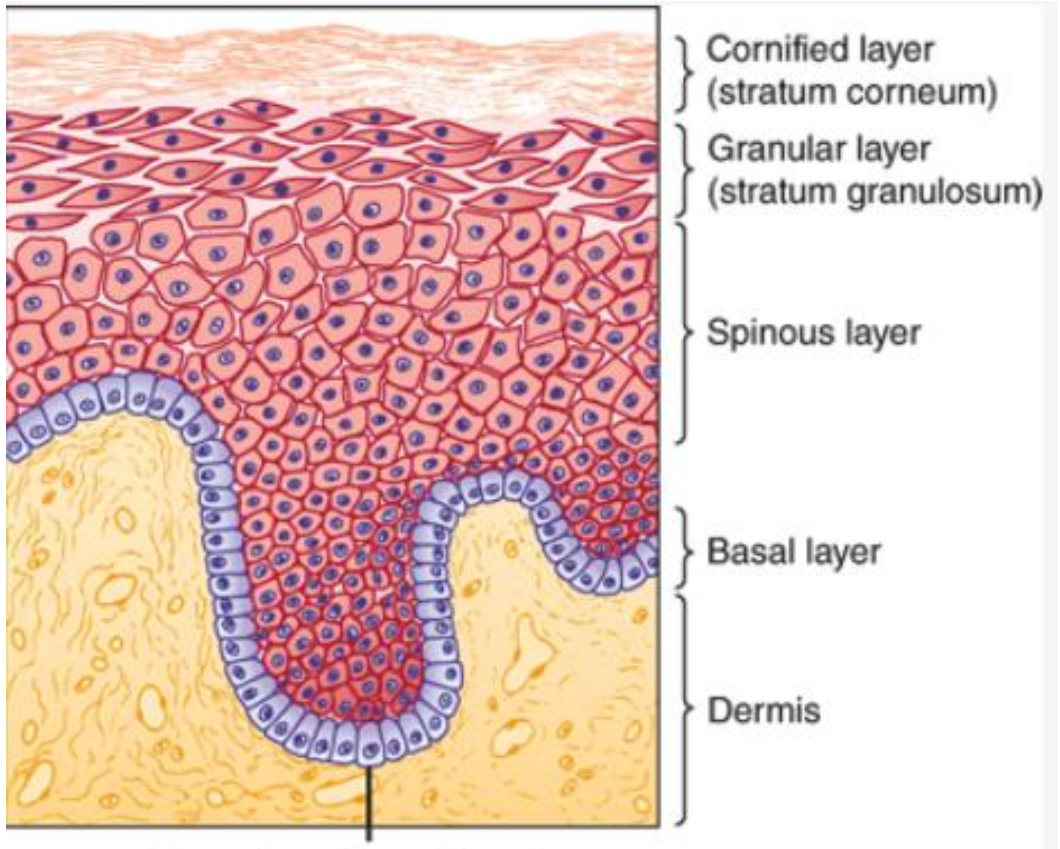
INTEGUMENTARY SYSTEM

- Protective Function
  - Barrier to disease (moisture in; pathogens out)

- Diseases of the skin
  - Neoplasm
  - Inflammatory
  - Infectious
  - Genetic cutaneous disorders

- Some are minor, others have significant morbidity consequences

ANATOMY



Layers of the skin

1. Epidermis
   - Stratified squamous epithelial
   - 4 layers (basal, spinous, granular, cornified)
2. Dermis
   - Connective tissue
   - Supports eccrine, and follicular structures
3. Subcutis
   - Adipose tissue

EVALUATION OF THE INTEGUMENTARY SYSTEM

Terminology:

Dermititis – inflammatory skin condition (i.e. rash)

Macule – flat area of discolouration without texture change (<1cm)

Patches – flat area of discolouration without texture change (>1cm)

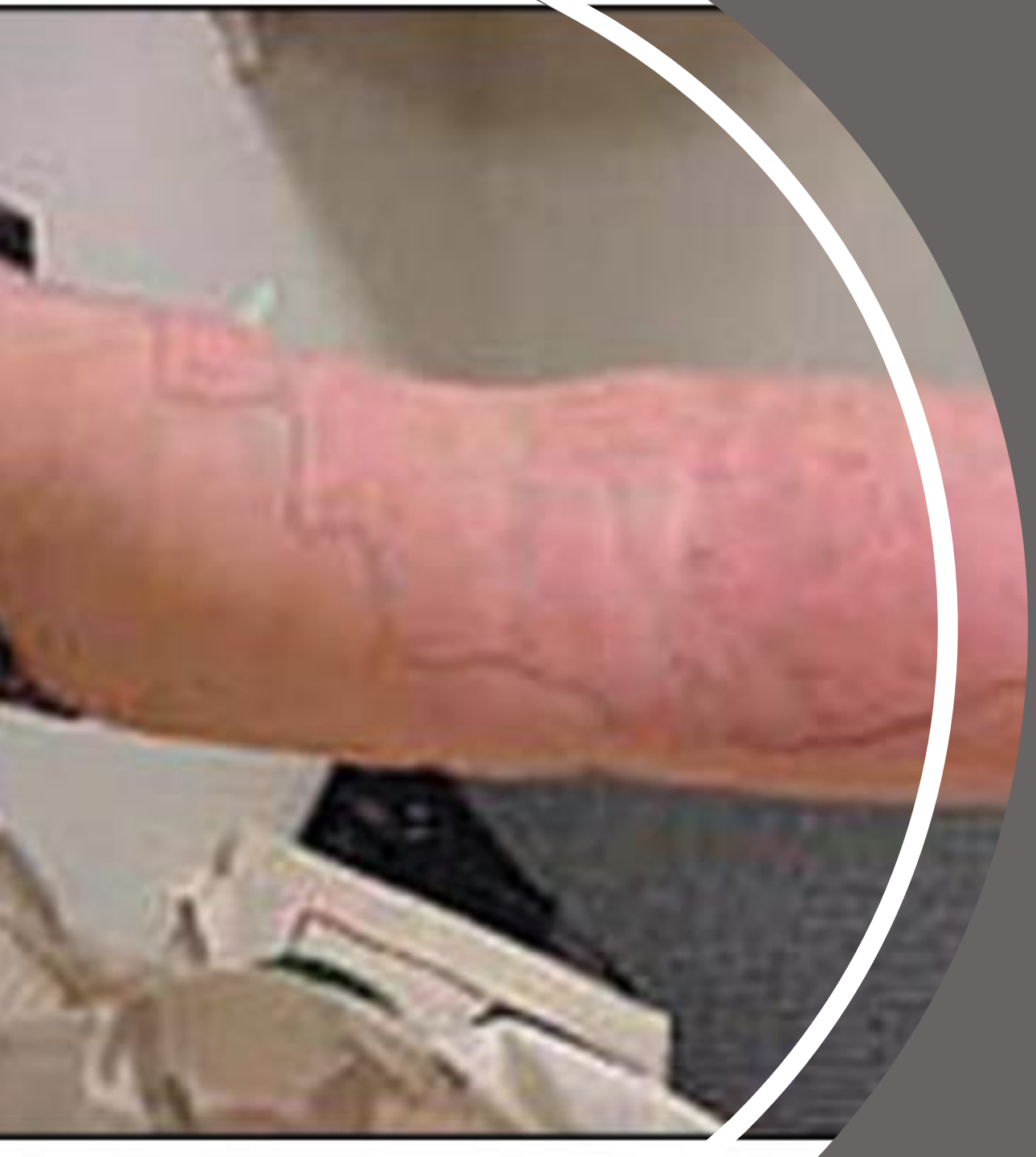
Papules – elevated, palpable skin lesions <1cm

Plaques – elevated, palpable skin lesions >1cm

Vesicles or bullae – fluid-filled spaces within the skin (dependent on size)

Pustule – vesicle or bulla contiaining purulent fluid

Nodule – solid, rounded skin lesion

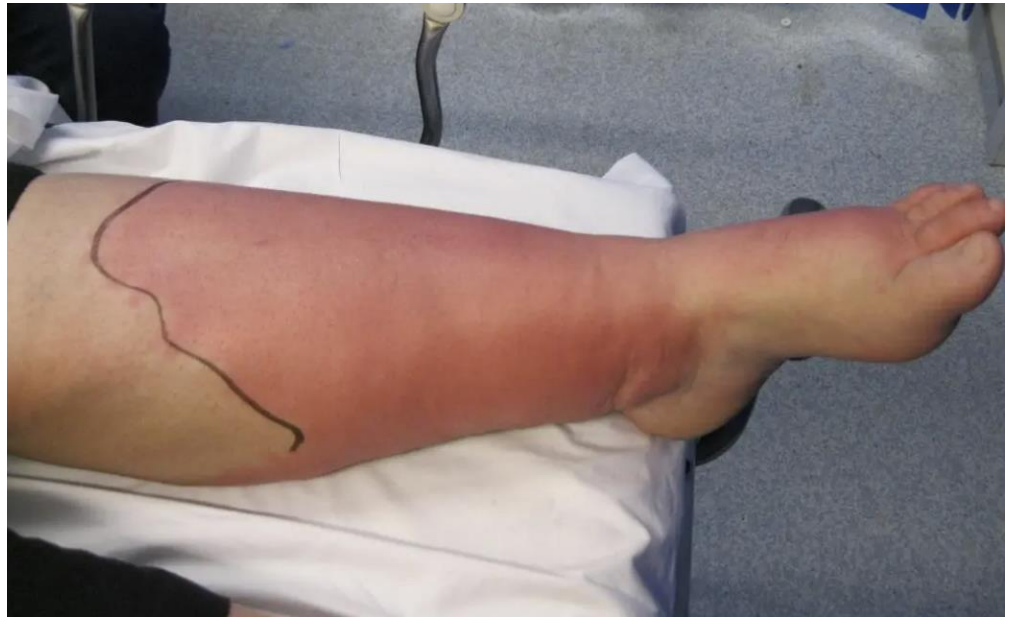


DISORDERS OF THE SKIN - INFECTIOUS

- Viral
  - Herpes
  - Verrucae
- Fungal
  - Superficial
  - Yeast
- Bacterial
  - Impetigo
  - Syphilis
  - Leprosy

CELLULITIS

- Bacterial skin infection
- Pain, redness, heat, swelling
- Common reason for fever and sepsis (especially in elderly)
- Very common presentation in paramedicine
- Always keep on your differential for source of fever!

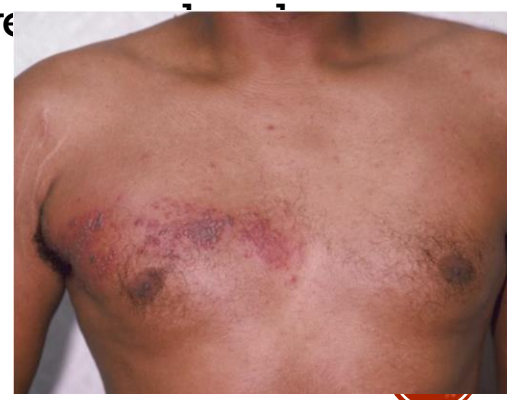


HERPES SIMPLEX VIRUS

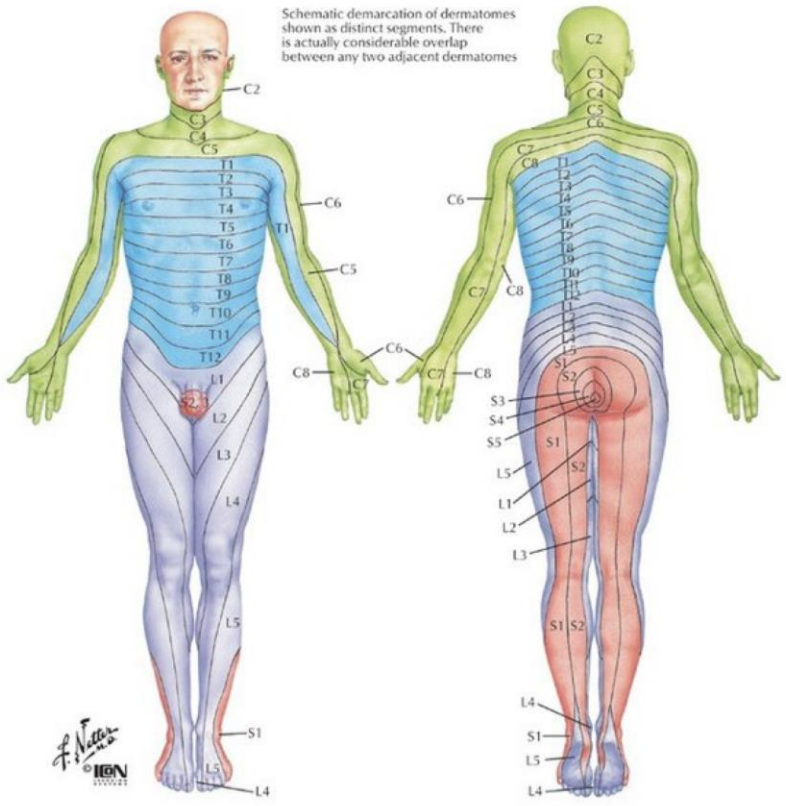
- Two Types: Type 1 and Type 2
- Mostly occurs above the waist
- HSV-1 may spread around other parts of the body through occupational hazards such as dentistry and medicine
- HSV-2 most infections in genital regions
- Begin with burning, tingling sensation
- Followed by vesicles, erythema and progress to pustules, ulcers, and crusts before healing
- Lesions most likely on lips, face, mouth
- Healing in 10 to 14 days
- Infection stays dormant in the trigeminal nerve and other ganglia
- Precipitated by: stress, sunlight, menses or injury
- Tx: no known cure. Pain management (lidocaine, Benadryl) and Acyclovir (antiviral) – shortens time of disease

HERPES ZOSTER

- Vacicella-zoster virus
  - Varicella (chickenpox)
  - Herpes zoster (shingles) from reactivation of latent VZV
- Clinical: painful, unilateral vesicular eruption, usually restricted to dermatomal distribution (in the case of shingles)
- Patho: infects nasopharyngeal lymphoid tissue through airborne droplets
- Inhibits multiple host defenses, enhancing infection
  - Prolonged incubation period prior to onset of skin lesions reflects time to ove
    immune defenses
- Rash – cell-free virus infects nerve endings (neuropathic pain)



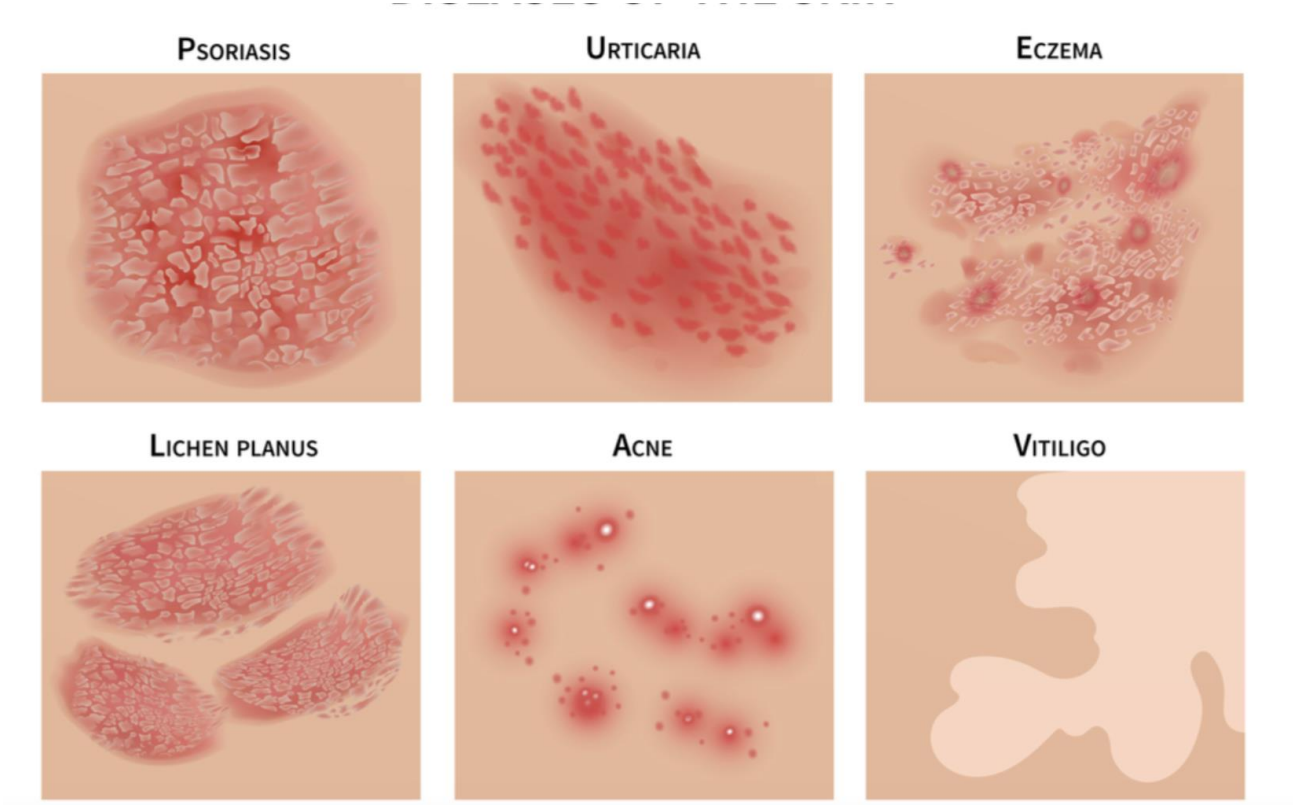
HERPES ZOSTER



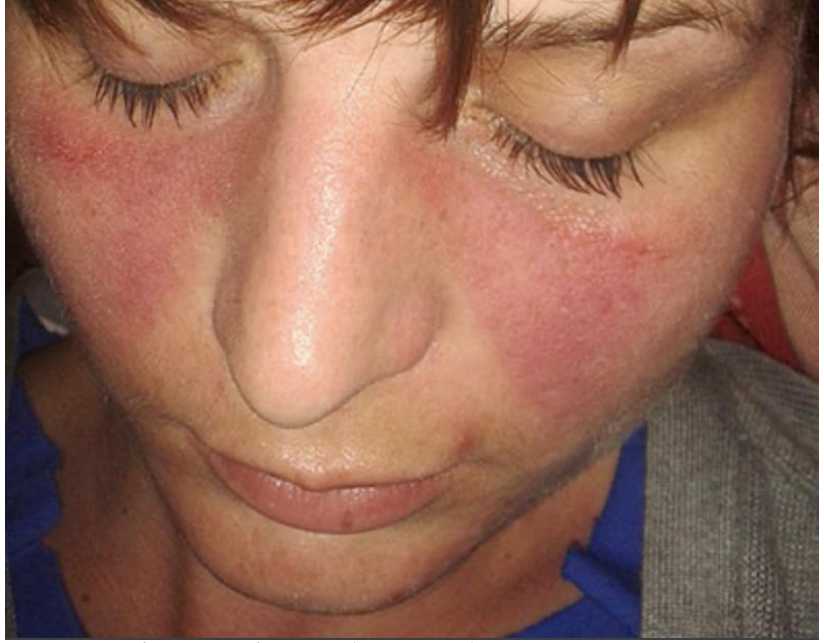
- CM: eruption of vesicles with erythematous bases restricted to skin supplied by sensory neurons or a single dorsal root ganglia
- Unilateral dermatomal distribution
- Most often thorax, trunk, and face
- Vesicles erupt for 3 to 5 days and usually clear up in 2 to 3 weeks
- Postherpetic neuralgia – complication occurring in people older than 50 years.
  - Pain that persists longer than the rash and blisters (can be months or years)

DISORDERS OF THE SKIN - INFLAMMATORY

- Lupus Erythematosus
- Seborrheic Dermatitis
- Psoriasis
- Lichen Planus
- Pityriasis Rosea
- Acne Vulgaris



LUPUS ERYTHEMATOSUS



- SLE is a chronic autoimmune disease of unknown cause
- Can impact any organ
- Immunologic abnormalities, esp. production of antinuclear antibodies (ANA) are a prominent feature
- Much of the damage results directly or indirectly from antibody formation and creation of immune complexes.
- Clinical presentation: mild join and skin involvement to life-threatening kidney, hematologic or CNS involvement.
- Patho: skin lesions are multifactorial
  - UV light as exposure – damages DNA – patient makes antibodies to DNA, IC form, complement is activated, local inflammatory response

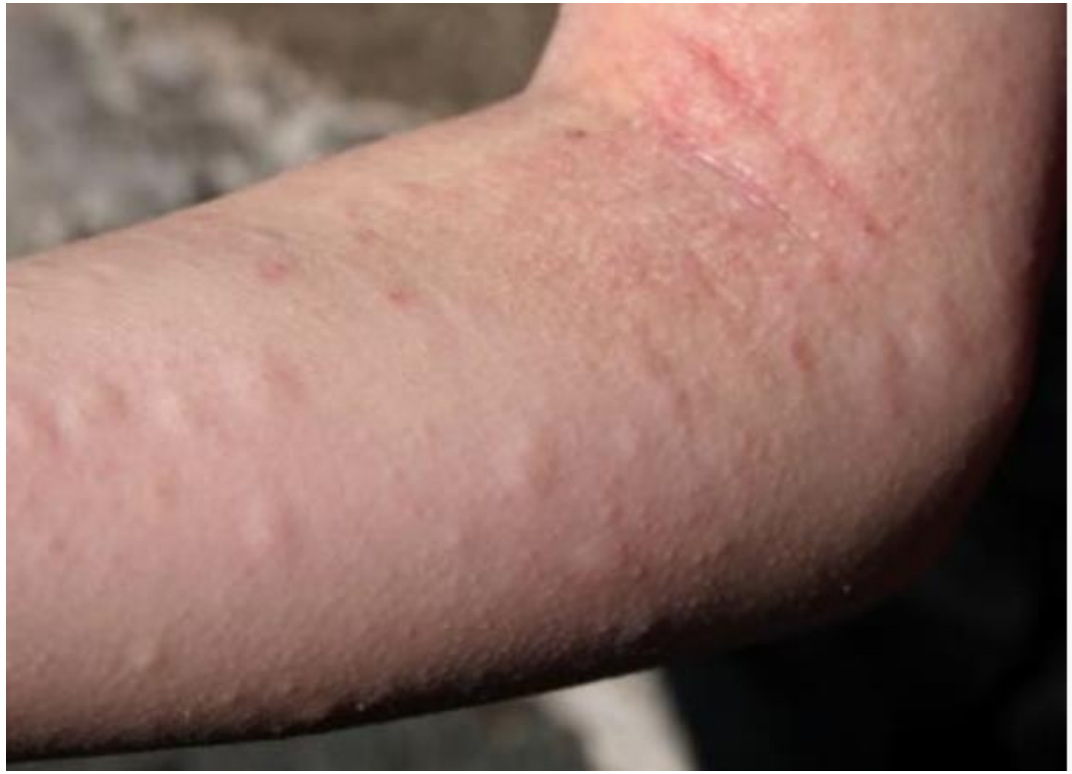
PSORIASIS

(PSORIASIFORM DERMATITIS)

- Chronic, persistent or relapsing, scaling skin condition
- Lesions are sharply marginated, erythematous, and surmounted by silvery scales
- Variation in presentation
- 1% to 2% of individuals; most common age of onset is 3rd decade (can develop whenever)
- Large genetic component, environmental factors (infection, physical injury, stress, drugs)
- Patho: inflammatory skin disease, epidermis is thickened and excessive epidermopoiesis (proliferation) – up to 30x as many keratinocytes per unit as normal skin
- Accumulation of cells in cornified layer with retained nuclei (due to truncation of cell cycle) – parakeratosis
  - Parakerototic cells accumulate, neutrophils migrate to cornified layer – resulting in the silvery scale of psoriatic plaques
  - Increased capillary permeability contributes to erythema
- Clinical:

DISORDERS OF THE SKIN – ALLERGIC RESPONSES

- Atopic dermatitis
- Contact dermatitis
- Drug eruptions
- Vasculitis
- Urticaria and Angioedema



URTICARIA

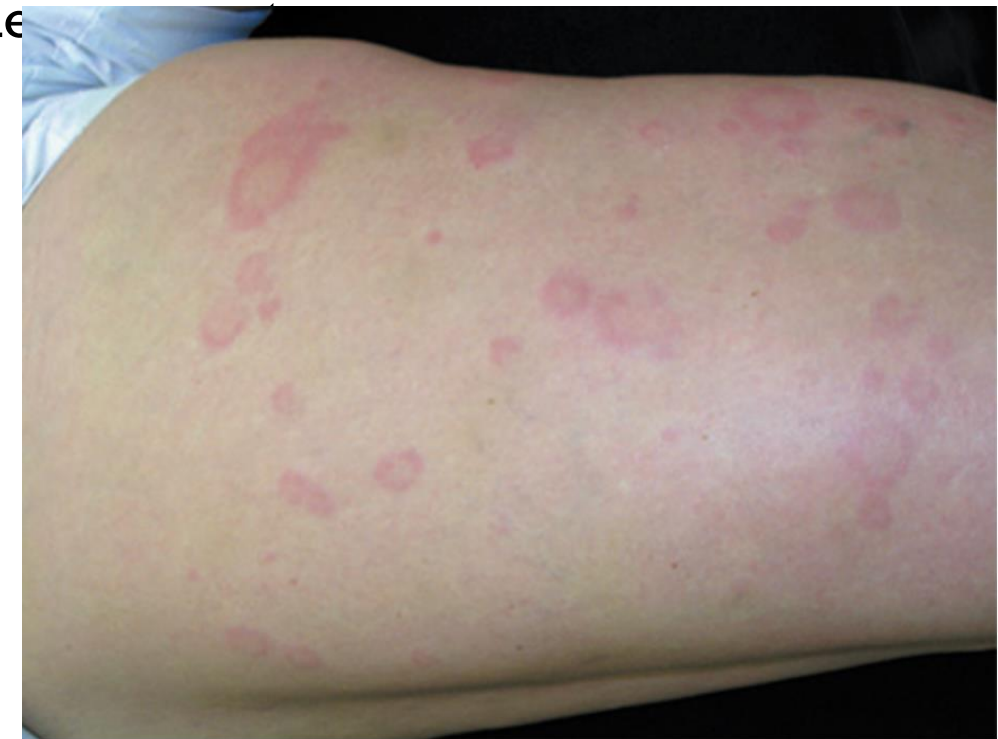
(PERIVASCULAR DERMATITIS)

- Clinical Presentation:
  - Transient papules and/or plaques ("hives" or "wheals")
  - Involves superficial dermis
- Can be acute (< 6 weeks), chronic (>6 weeks), or associated with acute allergic reactions (e.g. anaphylaxis)
- Often associated with angioedema
- Cause: sunlight, medication, pressure, vibration, heat, cold, exercise, emotional stress, food, insects, or no obvious trigger
- 20% of individuals at some point in life (11% to 35% of ED dermatologic presentations)
- Patho: perivascular infiltrate (lymphocytes, eosinophils, neutrophils)
  - Results from mast cell degranulation causing histamine release and proinflammatory cytokines (e.g. prostaglandins, leukotrienes, platelet activating factor)
  - Typically it is a Type I hypersensitivity reaction mediated by IgE
  - Histamine causes capillary vasodilation in superficial dermis, extravasation of protein-rich fluid into superficial skin – urticarial papules and/or plaques
  - Lesions resolve when fluid is reabsorbed

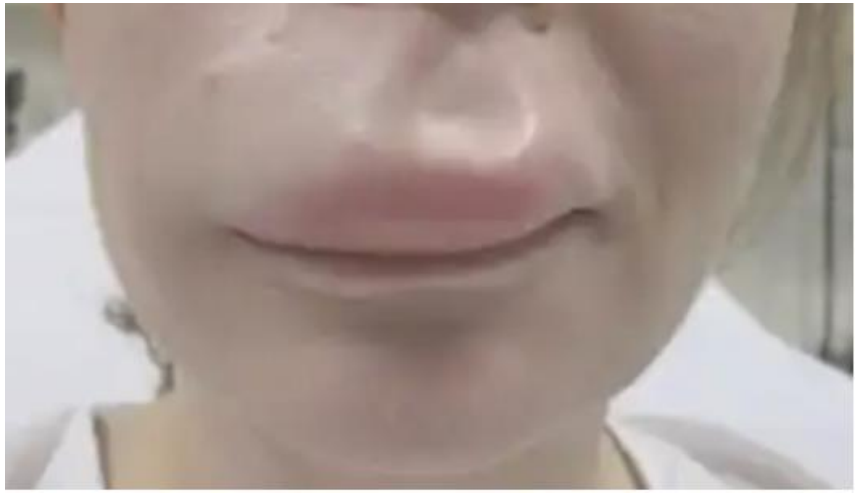
URTICARIA

(PERIVASCULAR DERMATITIS)

- Clinical Manifestations: papule or plaque without epidermal changes. Erythematous to white and range in size from mm to cm.
- Individual lesions are often pruritic
- Can involve any part of the body, trunk and extremitie
- Tx:
  - Avoid triggers
  - Avoid anti-inflammatory drugs
  - Antihistamines (diphenhydramine)



ANGIOEDEMA



- Similar process as urticaria
- Involves deep dermis and subcutaneous tissue
- typically presents as diffuse swelling rather than rash due to the deeper location of changes
- Most often presents around mucous membranes and or hands and feet
- Can be life-threatening if respiratory tract is involved

DISORDERS OF THE SKIN – PARASITIC INFECTIONS

- Scabies
- Fleas
- Lice
- Chiggers
- Bedbugs
- Ticks
  - Rocky Mountain Spotted Fever
  - Lyme Disease

DISORDERS OF THE DERMIS

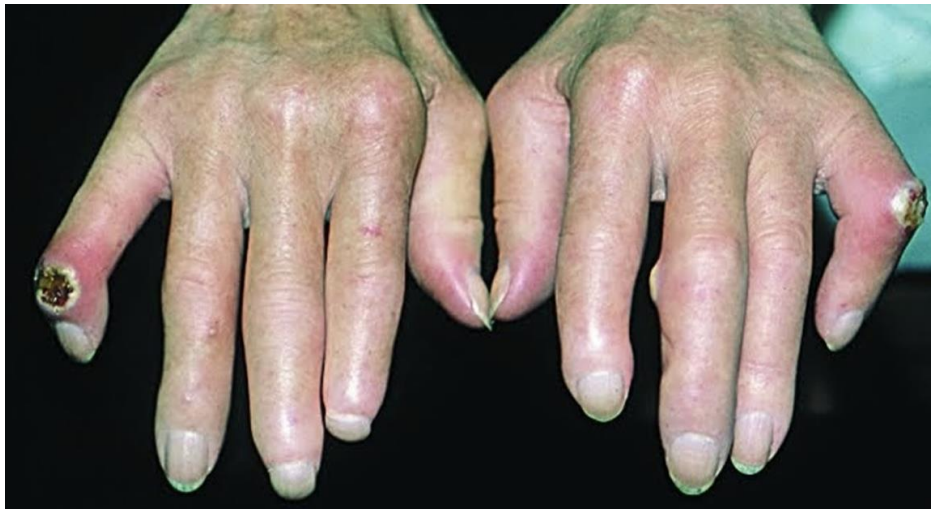
- Scleroderma

SCLERODERMA

- Presence of thickened, hardened skin
- Feature of systemic sclerosis (SSc)
  - Chronic multisystem disease – widespread vascular dysfunction and progressive fibrosis of the skin and internal ograns.
- Heterogeneous disease, broad range of organ involvement, disease severity and outcomes

SCLERODERMA

- Limited cutaneous systemic sclerosis – puffy fingers distal to metacarpophalangeal joints, skin sclerosis distal to elbows and knees
- Diffuse cutaneous systemic sclerosis – puffy hands and develop skin thickening that extends proximally to upper arms, thighs and/or trunk
- Clinical Features: pain and fatigue in addition to organ system involvement
  - Cutaneous – hardens and thickens; fingers, hands, face. Edematous swelling and erythema. Additional symptoms: pruritus, edema, loss of hair, dry skin
  - Vasculopathy –
  - MSk manifestations –
  - Gastrointestinal involvement –
  - Pulmonary involvement –
  - Cardiac involvement –
  - Renal involvement –
  - Neuromuscular involvement -



DISORDERS OF THE DERMIS

- Ulcers

ULCERS

- Open sore in skin or mucous membrane caused by poor blood flow
- Impact epidermis, dermis and even subcutaneous tissue
- Etiology: exposure to heat, cold, irritation, or problems with blood circulation (ie diabetes, pressure)

PRESSURE ULCERS

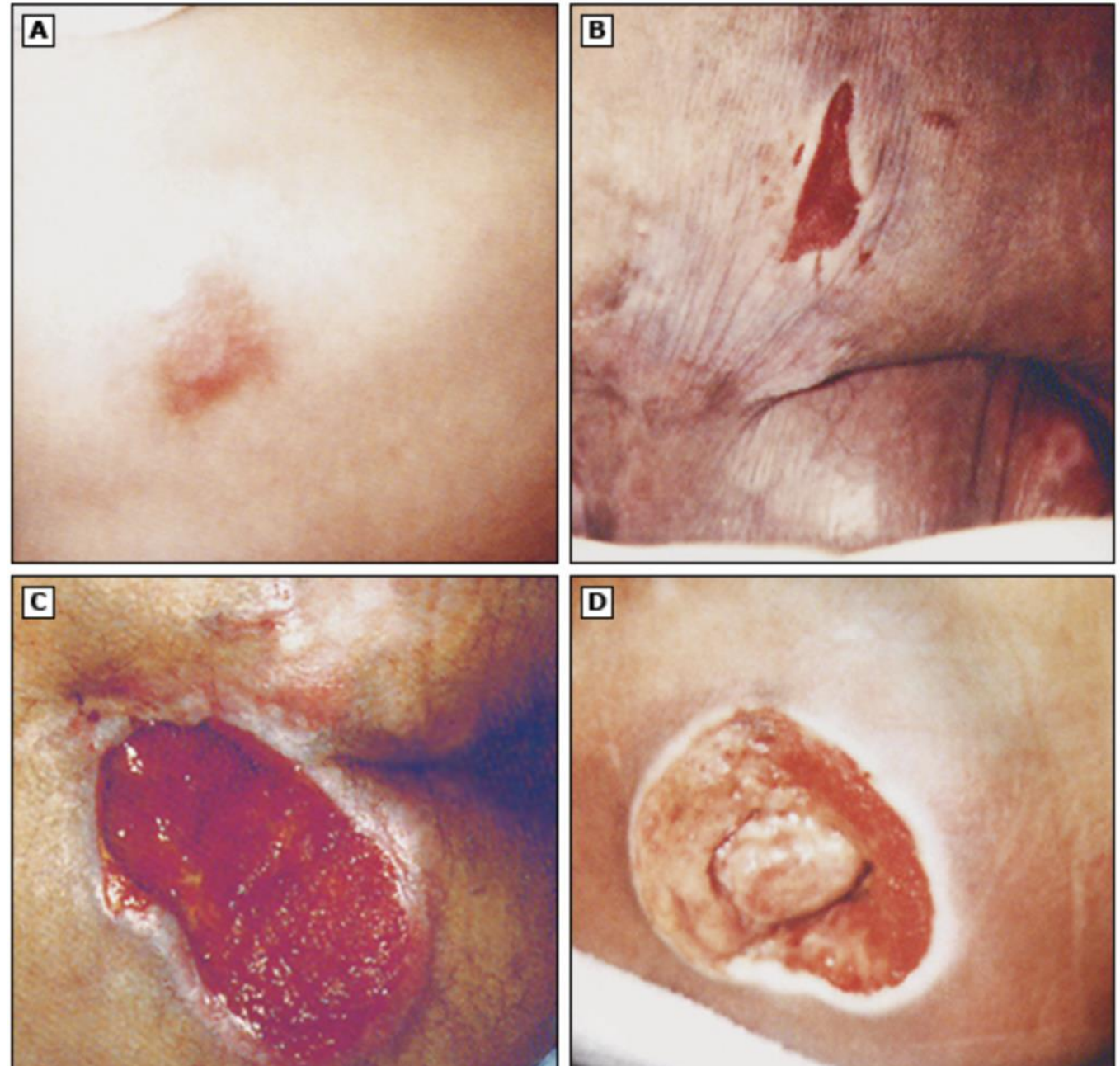
- Chronic pressure in a susceptible area leading to ischemia and skin loss
- Usually occur over bony prominence (sacrum, ischial, trochanteric wounds in wheelchair or bed-bound), heel ulcers
- Deeper tissues more susceptible than superficial skin (external appearance may underestimate the extent of damage)
- Patho: external factors + host-specific factors = tissue damage
  - Excess pressure (>32mmHg) prevents delivery of oxygen and nutrients to tissue – hypoxia, accumulation of met. wastes, free radical generation.
  - 1-4 hrs of sustained pressure load
- Over 100+ risk factors. Most important (immobility, malnutrition, reduced perfusion, sensory loss)

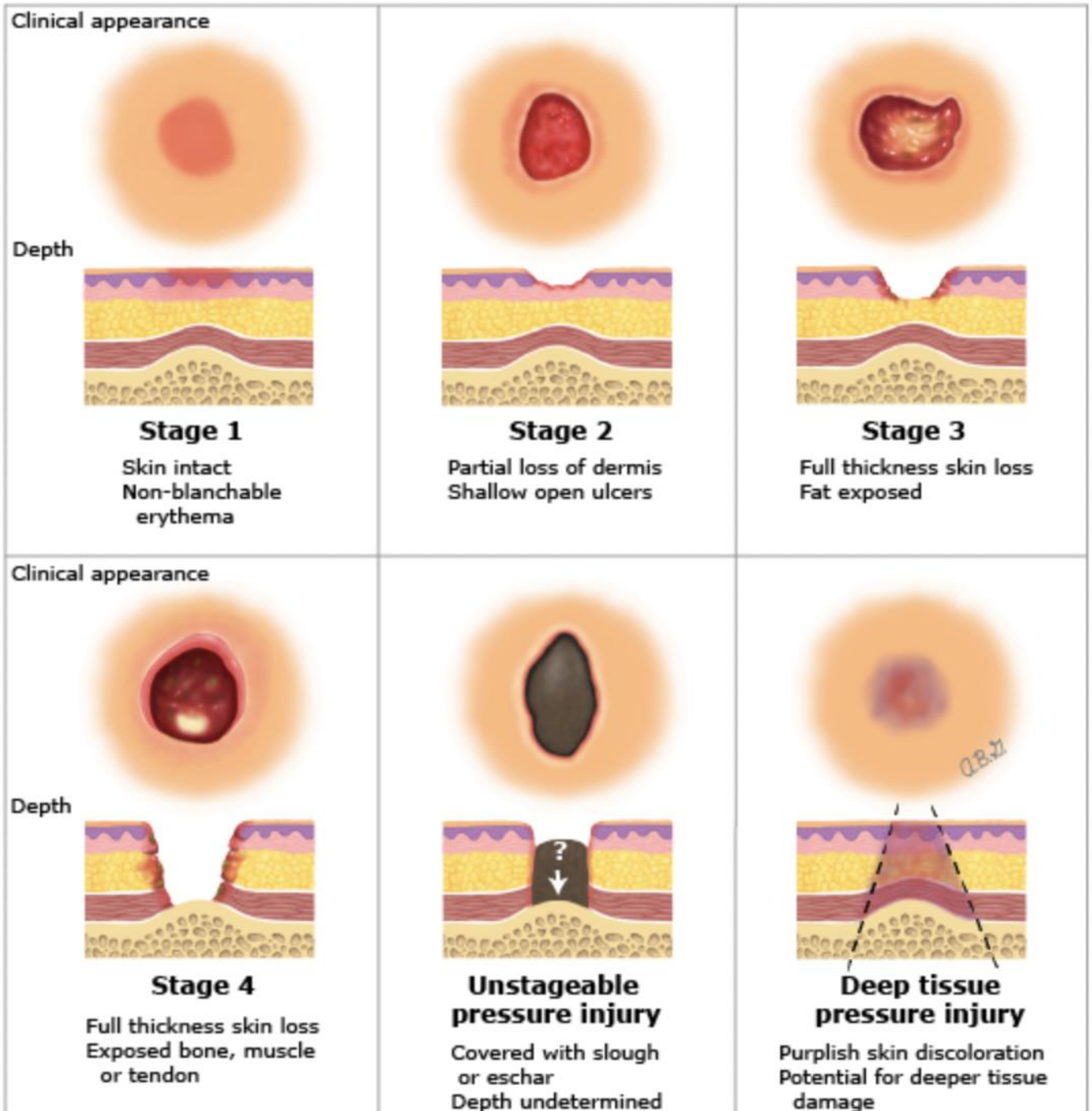
PRESSURE ULCERS

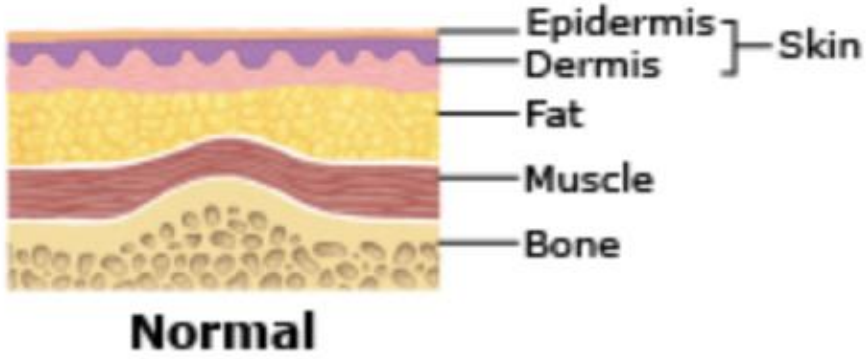
Stage	Description
1	Skin intact but with non-blanchable redness for >1 hour after relief of pressure.
2	Blister or other break in the dermis with partial thickness loss of dermis, with or without infection.
3	Full-thickness tissue loss. Subcutaneous fat may be visible; destruction extends into muscle with or without infection. Undermining and tunneling may be present.
4	Full-thickness skin loss with involvement of bone, tendon, or joint, with or without infection. Often includes undermining and tunneling.
Unstageable	Full-thickness tissue loss in which the base of the ulcer is covered by slough and/or eschar in the wound bed.
Deep tissue pressure injury	Purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying tissue from pressure and/or shear.

Reference:

1. *National Pressure Injury Advisory Panel. NPIAP Pressure Injury Stages. Available at: https://npiap.com/page/PressureInjuryStages (Accessed on January 29, 2020).*


A
B
C
D


Clinical appearance
Depth
Stage 1
Skin intact
Non-blanchable erythema
Stage 2
Partial loss of dermis
Shallow open ulcers
Stage 3
Full thickness skin loss
Fat exposed
Clinical appearance
Depth
Stage 4
Full thickness skin loss
Exposed bone, muscle or tendon
?
Unstageable pressure injury
Covered with slough or eschar
Depth undetermined
Deep tissue pressure injury
Purplish skin discoloration
Potential for deeper tissue damage


Epidermis
Dermis
Skin
Fat
Muscle
Bone
Normal

Review > J Trauma Acute Care Surg. 2014 Apr;76(4):1131-41.
doi: 10.1097/TA.0000000000000153.

Pressure ulcers from spinal immobilization in trauma patients: a systematic review

Wietske Ham [1], Lisette Schoonhoven, Marieke J Schuurmans, Luke P H Leenen

Affiliations + expand

PMID: 24662882 DOI: 10.1097/TA.0000000000000153

Conclusion: The results from this systematic review show that immobilization with devices increases the risk for PU development. This risk is demonstrated in nine experimental studies with healthy volunteers and in four clinical studies.

DIABETIC ULCERS

- Foot ulcer, most frequent lower body complication
- Annual incidence 2.2%
- Up to 15% of diabetics have ulcers; 80,000 amputations in US
- More than ½ become infected
- Caused by repetitive stress and vertical or shear stress in an area with peripheral neuropathy
- Patho: combination of loss of glycemic control, peripheral neuropathy, peripheral vascular disease, immunosuppression
- Hyperglycemia produces oxidative stress on nerve cells and ischemia through glycosylation of nerve cell protein (neuropathy)
  - Leads to motor, autonomic, sensory complications
  - Imbalance in muscles, deformities, skin ulcerations
- Hyperglycemia leads to endothelial dysfunction and reduction of vasodilators, increase thromboxane A2 – leads to vasoconstriction and hypercoagulation – ischemia
- Hyperglycemia – increased T lymphocyte apoptosis - reduced healing response.

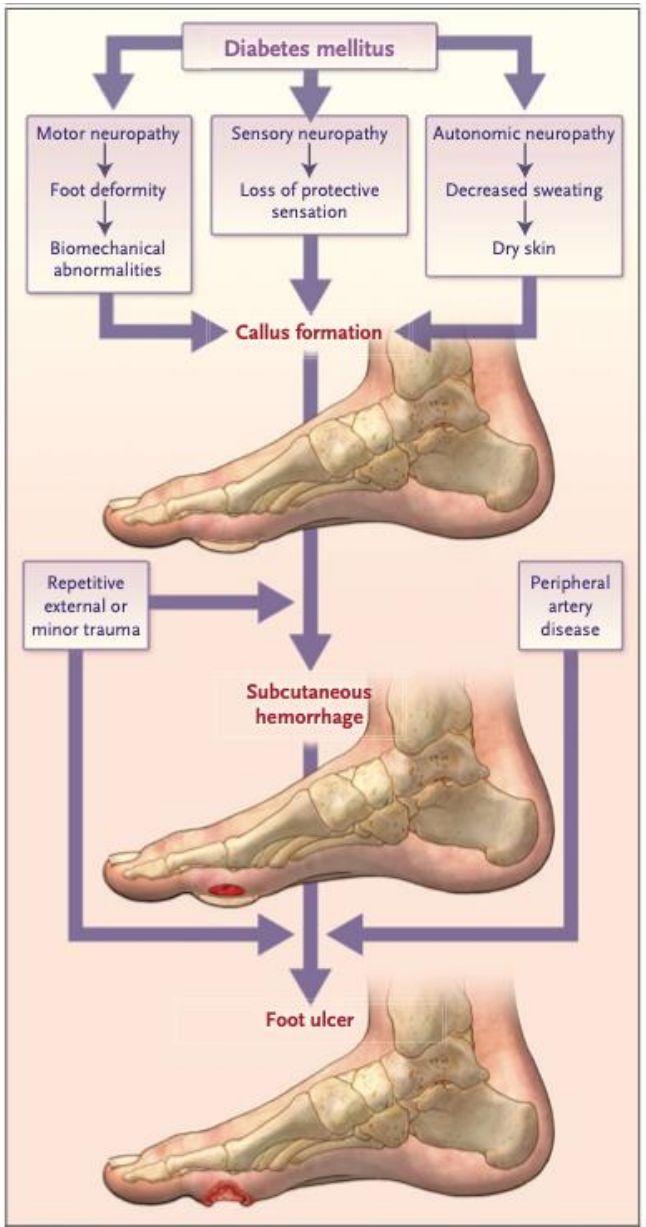
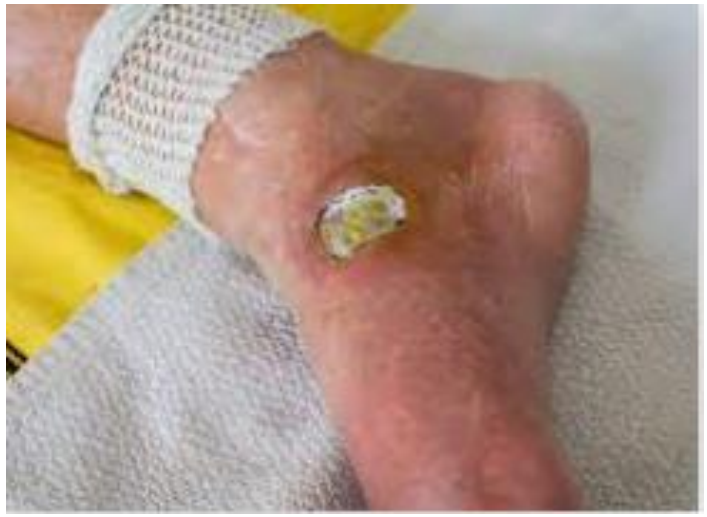


Figure 1. Common Pathway of Diabetic Foot Ulcer Occurrence and Recurrence.
Diabetic foot ulcers and their recurrences are caused by a number of factors that ultimately lead to skin breakdown. These factors include sequelae related to sensory, autonomic, and motor neuropathies.

DIABETIC ULCERS



- Tx: surgical debridement, offloading of pressure, attention to infection and potentially vascular reconstruction – foot ulcers can heal
- 40% reoccurrence in 1 year

- Interesting fact: risk of death at 5-years is 2.5x in diabetics with foot ulcers compared to no foot ulcers

BURNS

1. Electrical
   - AC more dangerous than DC
2. Thermal
   - Heat source, liquid (scald), energy transfer, hot objects, steam, cold objects
3. Chemical
   - Acid – coagulation necrosis
   - Alkaline – liquefaction necrosis
4. Radiation (sunburn, x-ray, nuclear)

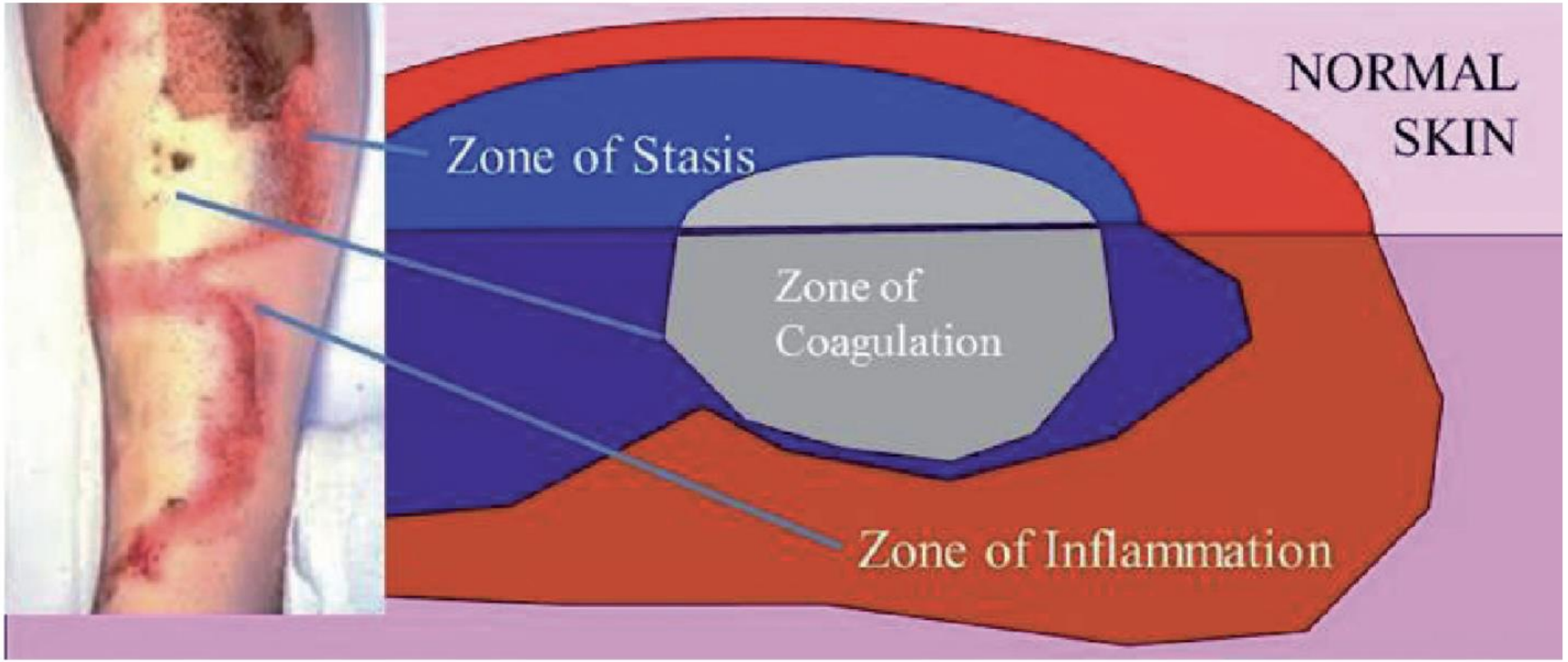
BURNS - EPIDEMIOLOGY

- 500,000 burns in US annually
  - 10% hospitalized
  - Majority present to ED
  - 3200 deaths
- 70% male, highest age group risk is 18-35 years
- 1/3 hospitalized burn patients with inhalation injury develop major upper airway obstruction

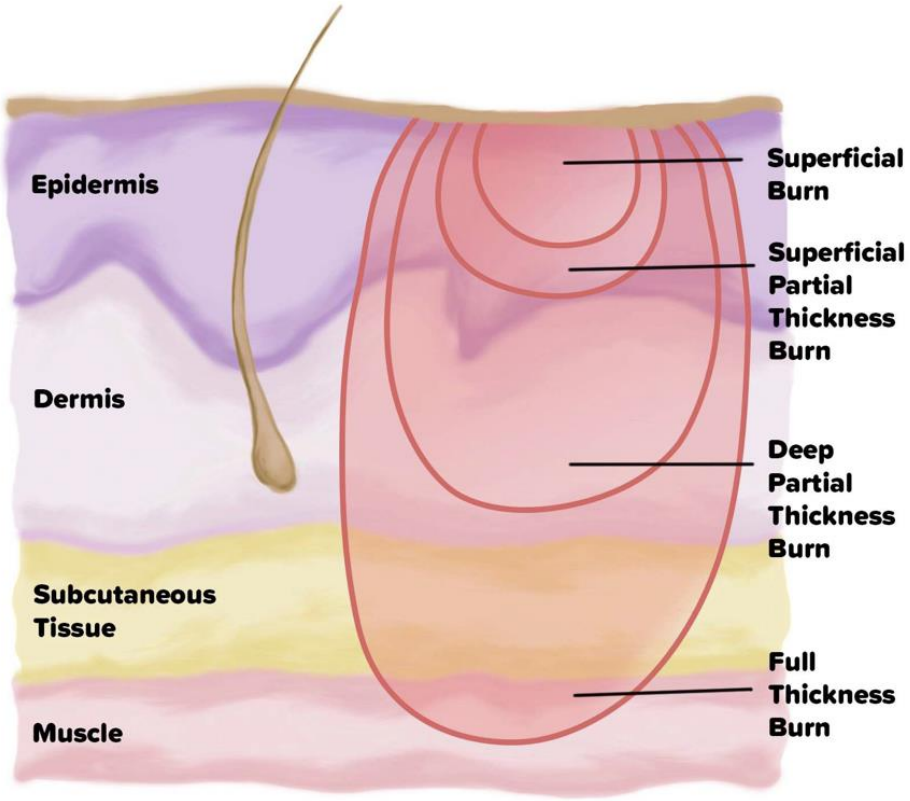
BURNS

- Disruption of homeostatic function of skin (temp regulation, fluid balance)
- Disruption of cell membrane causes water and electrolyte shifts, release of vasoactive substances (histamine, kinins, oxygen free radicals)

- Larger burns decrease cardiac output, metabolic acidosis, increase blood viscosity and hematocrit, anemia
- Classified based on severity, depth, size of burns

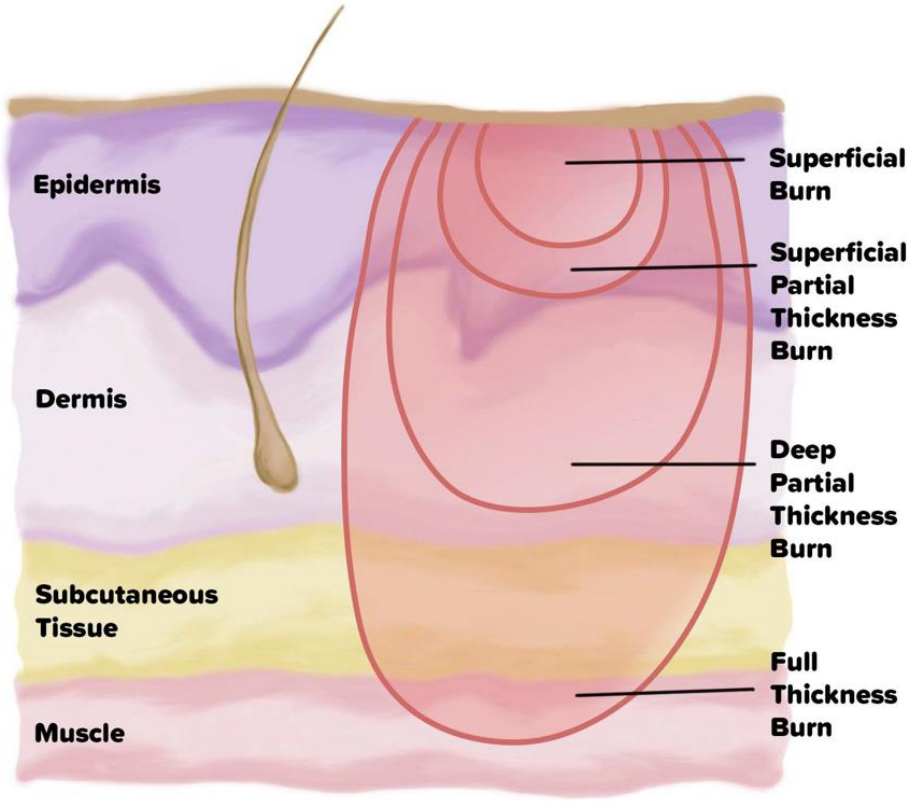

NORMAL
SKIN
Zone of Stasis
Zone of
Coagulation
Zone of Inflammation

BURNS - CLASSIFICATIONS



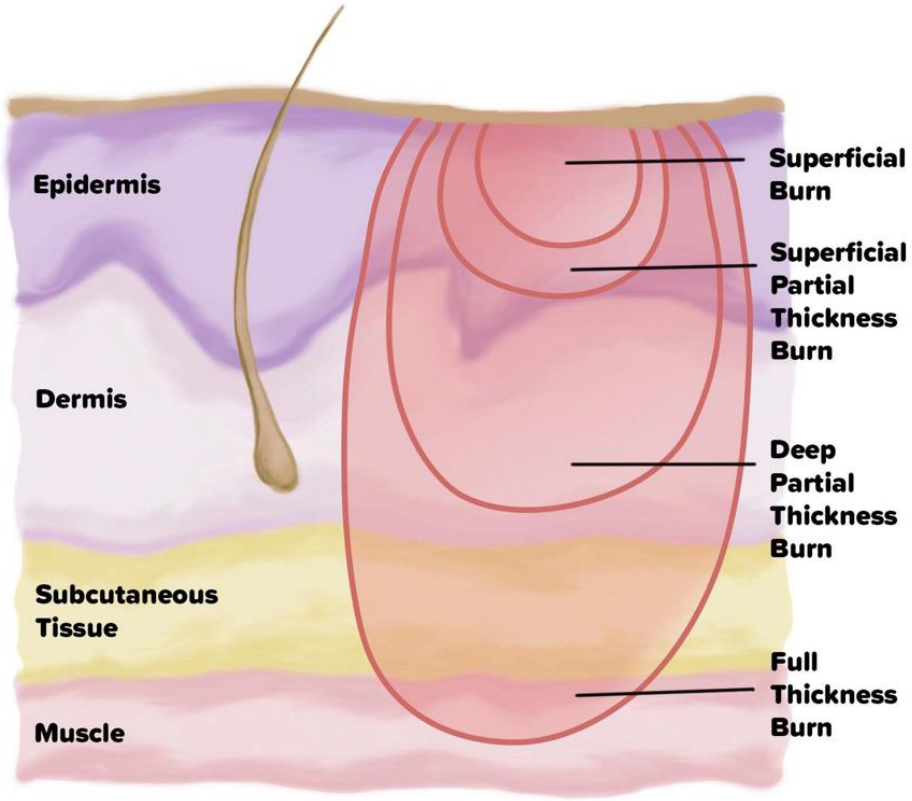
- Superficial Thickness (use to be called first degree)
  - Epidermis only
  - Redness, pain, no blisters
  - E.g. mild sunburn

BURN CLASSIFICATIONS

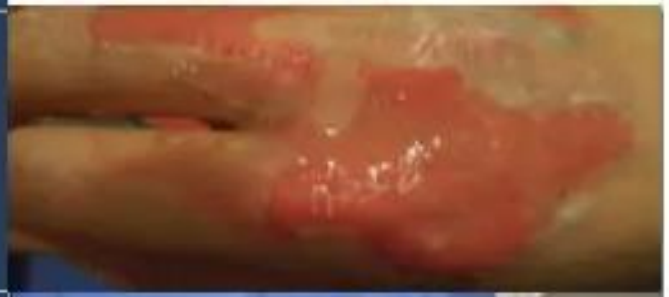
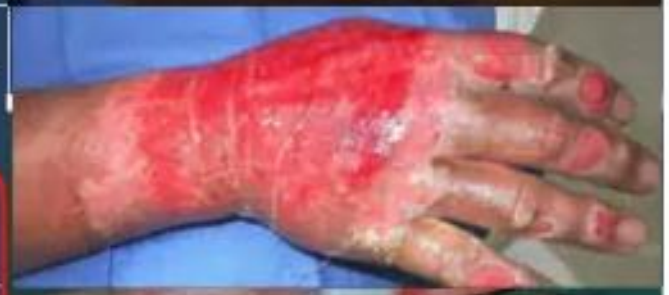
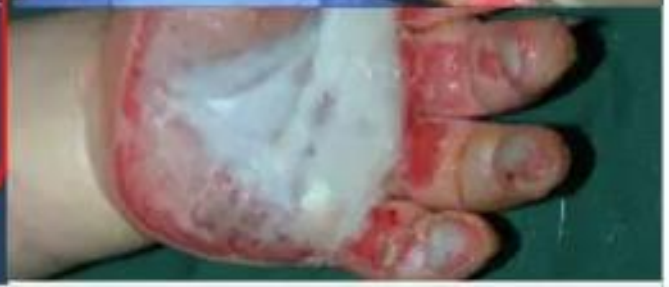


- Partial Thickness (use to e second degree)
  - Epidermis and part of dermis
  - A) Superficial partial thickness
    - Superficial layer of dermis
    - Erythematous because dermal tissue is inflamed.
    - Area blanches with pressure
  - B) Deep partial thickness
    - Deeper into dermis
    - Damage to hair follicles and glandular tissue
    - Painful, form blisters, wet or dry, waxy, may appear white

BURNS - CLASSIFICATIONS



- Full Thickness (use to be called third degree)
  - Through the full dermis, often to underlying subcutaneous tissue
  - Waxy white to leathery grey to charred and black
  - Not painful (damage to nerve ending), surrounding tissue is painful
  - Dead and denatured skin (eschar) is removed to aid healing (escharotomy)
  - Subdermal or Fourth degree burns – injury to deeper tissues such as muscle or bone
    - Blackened and leads to loss of burned part

BURN DESCRIPTION	APPEARANCE	CAP REFILL	SENSATION/ PAIN	HEALING	
1st SUPERFICIAL THICKNESS	ERYTHEMA	FAST	+	7-14D	
SUPERFICIAL PARTIAL THICKNESS 2nd	WET, PINK, BLISTERS,	FAST	++	2-4 WEEKS	
DEEP PARTIAL THICKNESS	LESS WET, RED, +/-BLISTERS,	SLUGGISH OR ABSENT	+/-	3-8WKS WITH SEVERE SCARRING; NEEDS **GRAFTING**	
3rd FULL THICKNESS	DRY, WHITE	ABSENT	ABSENT	NEEDS **GRAFTING**	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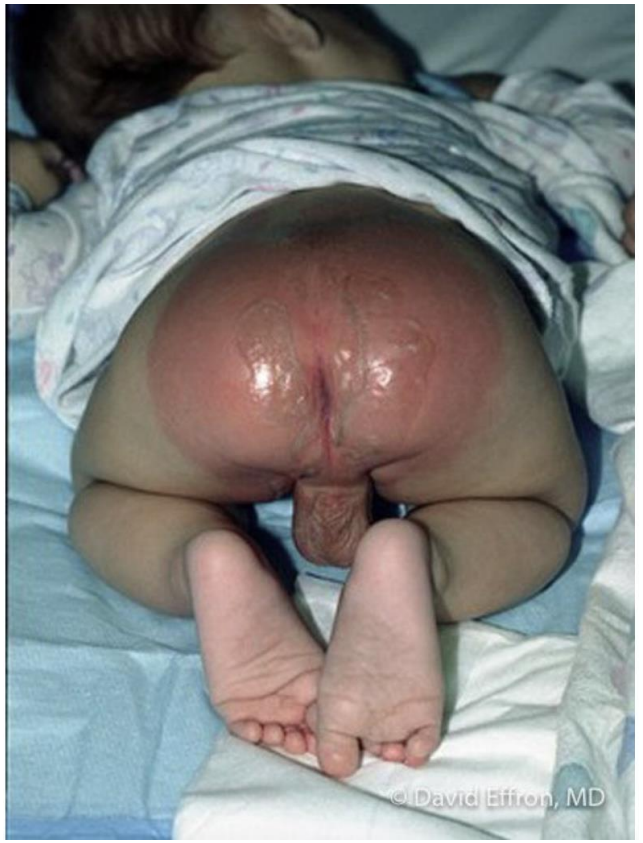
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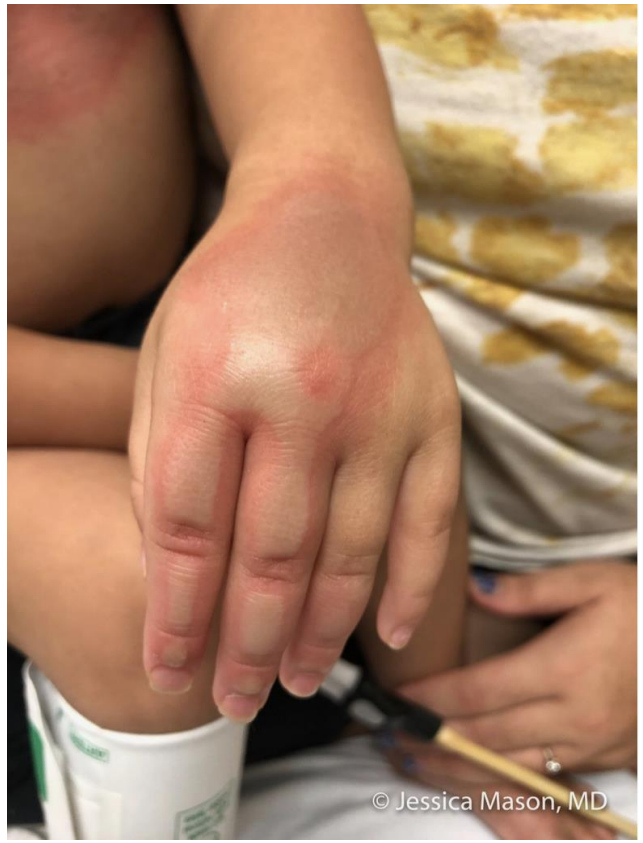
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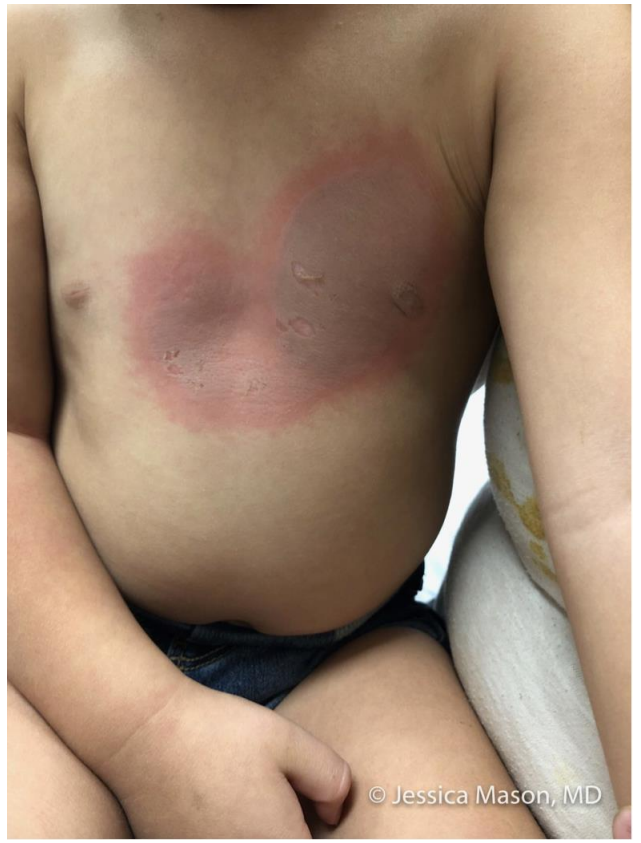
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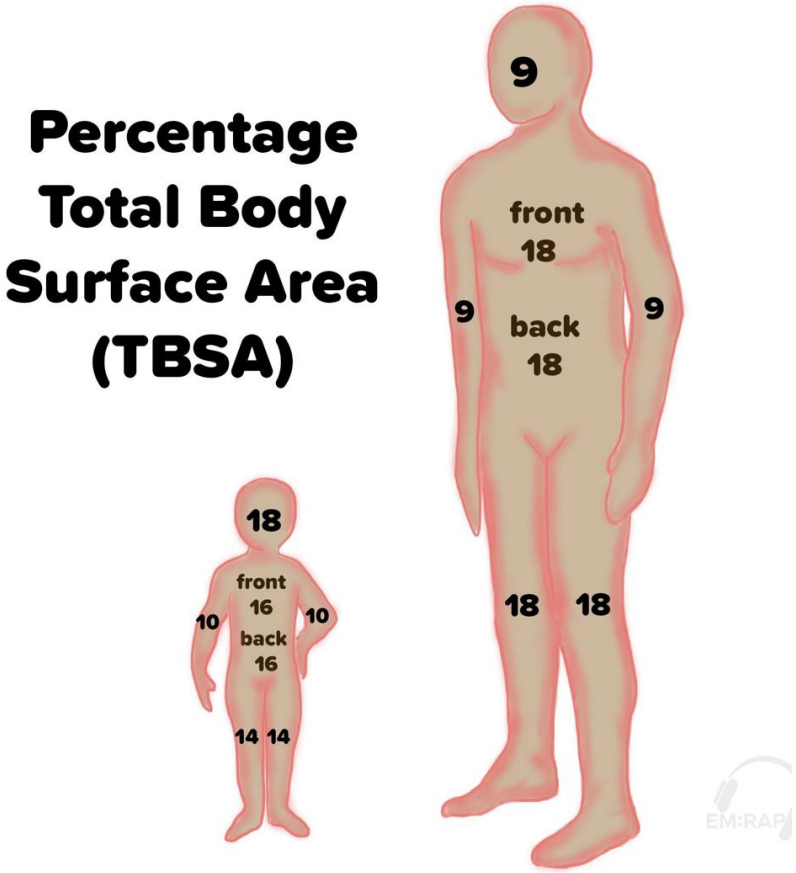
BURN ESTIMATION

- Rules of nines
- Rule of palms
- Lund and Browder



CHEMICAL BURNS

- Industrial accidents or household chemicals
- Alkali penetrate deeper and cause "worse" burns than acids
- Patho: penetrate the stratum corneum layer of skin through chemical reaction (not thermal)
- Acids - coagulation necrosis and protein precipitation – forms eschar, limits damage
- Alkali – liquefaction necrosis – deep penetration
- Certain agents require specific treatments

Agent	Treatment
Hydrofloric Acid	10% calcium gluconate
Chromic Acid	Sodium Hyposulphite
Dichromate Salts	Sodium Hyposulphite

ELECTRICAL BURNS

- Factors that predict injury
  - Voltage, AC or DC, Duration of contact, degree of wetness/humidity in environment

INTEGUMENTARY MANIFESTATIONS OF SYSTEMIC DISEASE

- Colour
  - Presence of systemic disease
  - Red (erythema), orange (carotene deposition), yellow (jaundice), green (severe jaundice – biliverdin), blue (cyanosis or Raynaud disease), violet (vascular insufficiency, or shock)
- Sensation
  - Pruritus
- Texture
  - Dehydration, edema
- Temperature
  - Fever, sweating, thermoregulation