Trauma and skin injury

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Outlines

- Burns
- Wound healing

B4.2.3 Traumatic and mechanical disorders

กลุ่มที่ 1 และ 2	กลุ่มที่ 3
(1) Burns	(1) Keloid, hypertrophic scar
(3) Scar	
(4) Ulcers	
(5) Wound	

Learning Outcomes

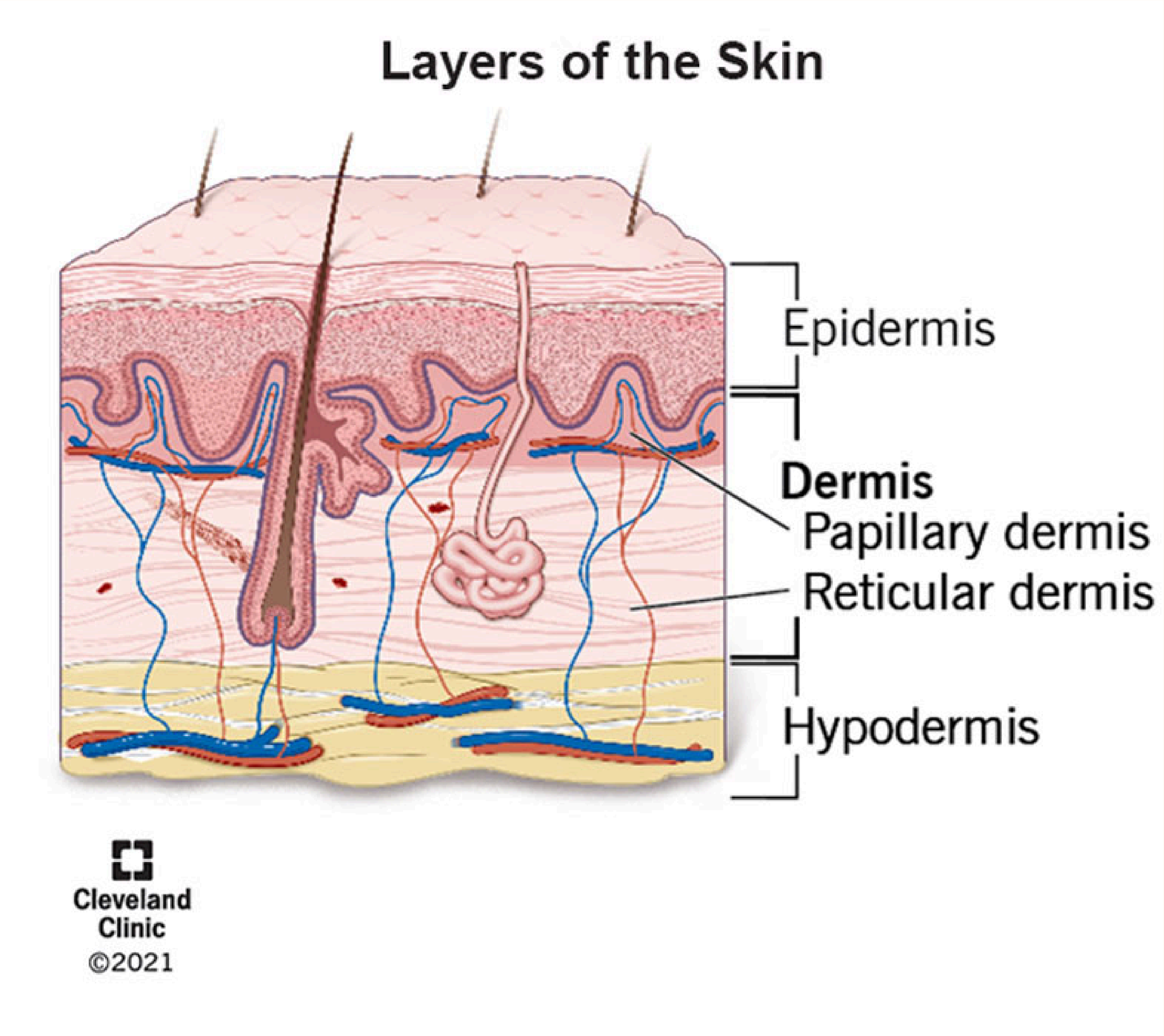
At the end of this lesson, you'll be able to describe:

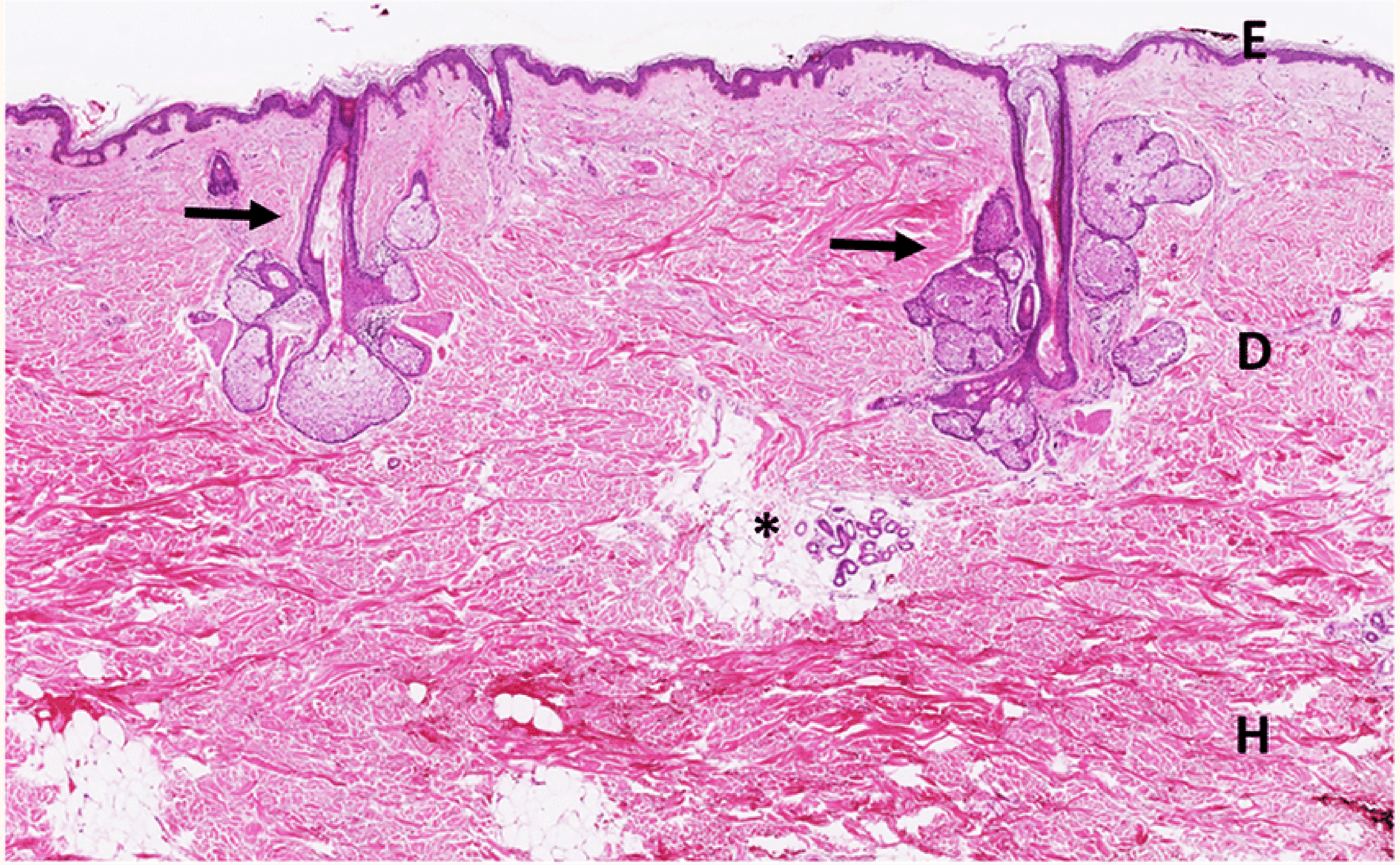
Burn
- Pathophysiology
- Types of burns
- Evaluation of burn severity
- Complications
- Initial treatment and management

Wound healing
- Pathophysiology
- Abnormalities in tissue repair

Layers of skin

EPIDERMIS	DEMIS	HYPODERMIS/ SUBCUTANEOUS TISSUE
• The intact keratinized epidermis serving as a mechanical barrier • Protecting from infection and fluid evaporation	• Made up of collagen fibers and where nerves, blood vessels, and skin appendage (sweat glands, sebaceous gland and hair follicles) reside	• Adipose tissue, temperature regulation


Layers of the Skin
Epidermis
Dermis
Papillary dermis
Reticular dermis
Hypodermis
Cleveland
Clinic
©2021


E
D
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*

skin has an important role to play in

Fluid and temperature regulation of the body

A protective barrier against bacteria

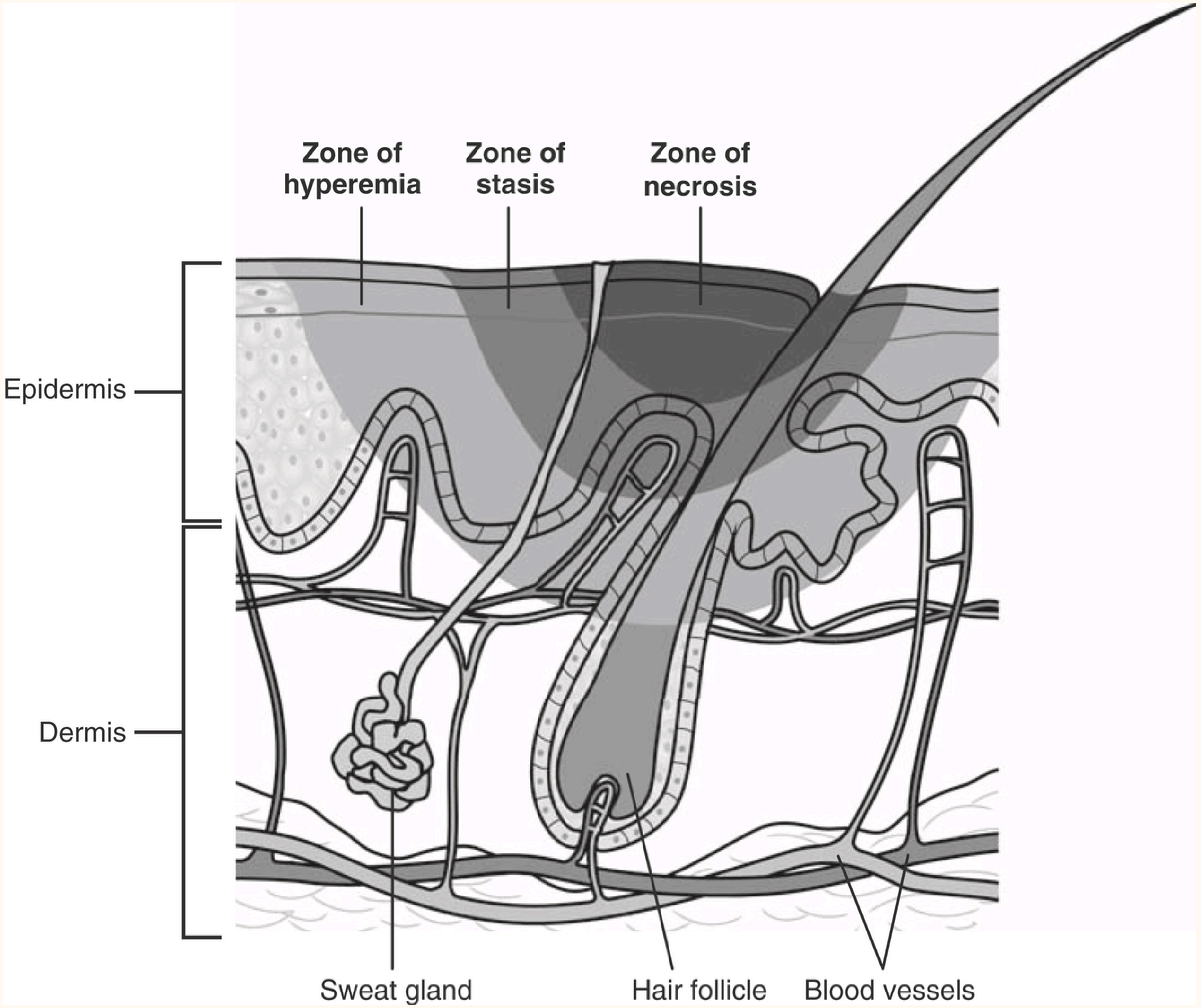
Immune mechanism

Sensation of pain, temperature and touch

BURN

Pathophysiology

- The injuries that result from burns are due to a combination of **immediate tissue injury and resulting inflammation.**
- Inflammation includes the activation of an inflammatory cascade, immediate and delayed cellular and humoral effects, and the delayed effects of changes in gene expression induced by the earlier injuries.
- The immediate injury is classically divided into **three zones**.
  a. **Central zone of necrosis** (the most severely injured)
  b. **Zone of stasis**
  c. **Zone of hyperemia**



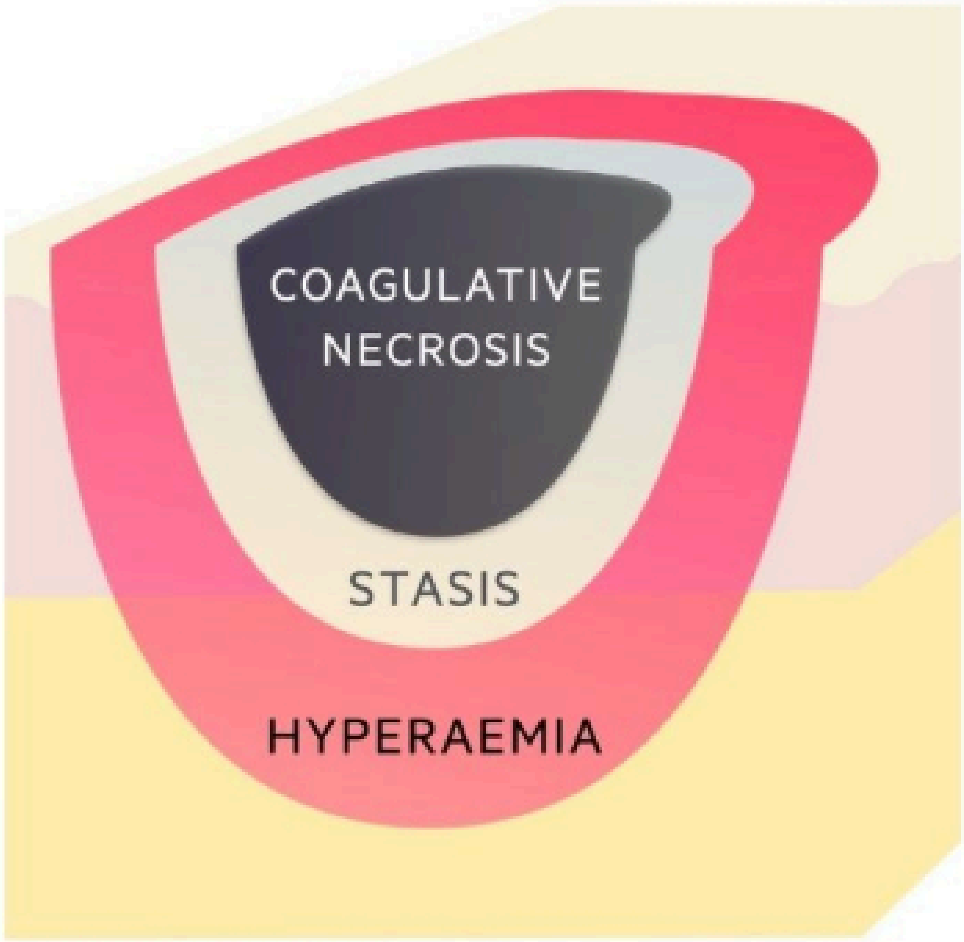
Pathophysiology

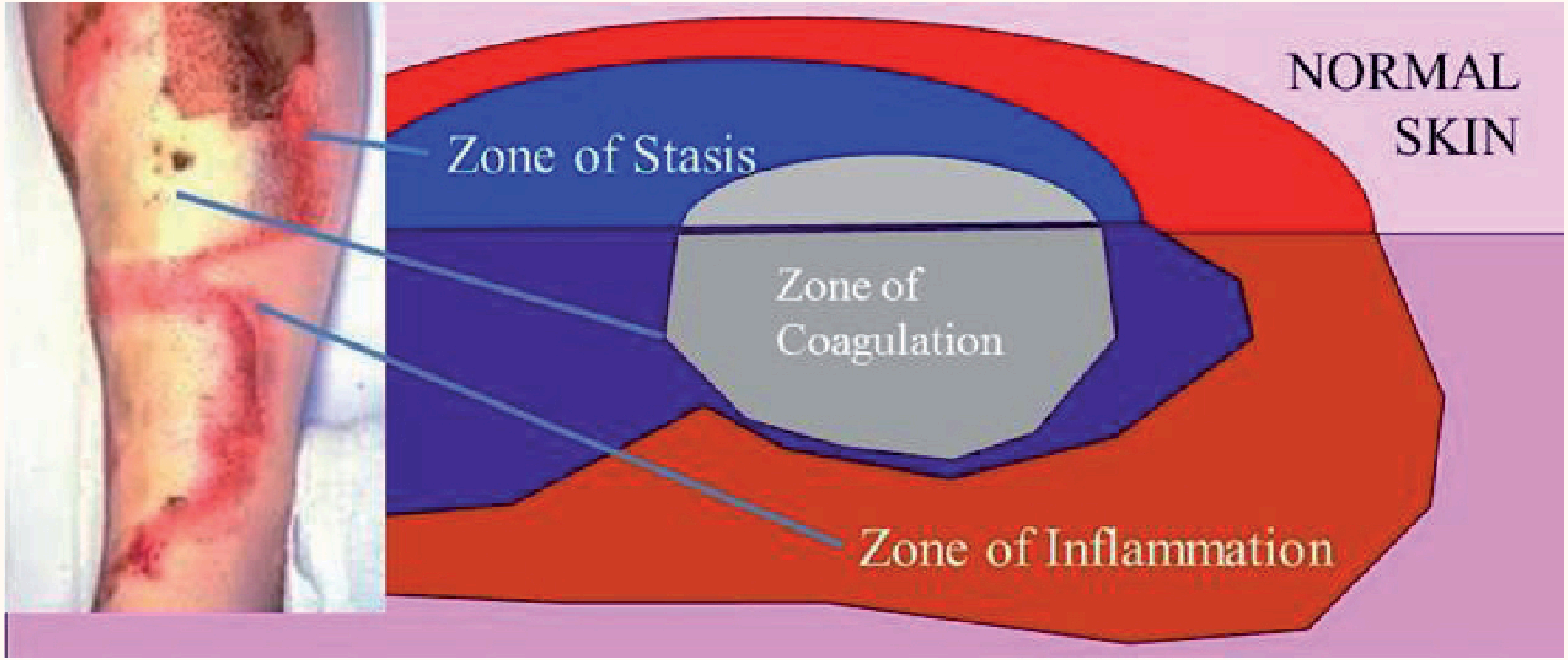
- The zone of necrosis is composed of **coagulated tissue** that will progress to rapid **cell death and eschar formation**.
- The outer zone of hyperemia has **increased blood flow and minimal tissue damage** and will almost certainly heal.
- The middle zone of stasis is the **region where blood flow has ceased within the capillary bed**.
- Depending on subsequent actions over the next 24 to 48 hours, this area either will worsen to necrotic tissue or may re-epithelialize to healing.
- The immediate postinjury efforts are focused on salvaging this area.

JACKSON'S THREE ZONES OF BURNS

WWW.OPENMED.CO.IN

ZONES	MECHANISM	TISSUE VIABILITY
ZONE OF COAGULATION	Direct Injury to Cells	Non Viable (Necrosed)
ZONE OF STASIS	Vascular Damage and Vessel Leak	May / May NOT be Viable
ZONE OF HYPEREMIA	Vasodilation	Viable (Healing Begins from here.)




NORMAL
SKIN
Zone of Stasis
Zone of
Coagulation
Zone of Inflammation

Burning tissue

- As heat transfers to tissue → damage begins to occur as cellular temperature rises above 44°C
- Cell die when internal cellular temperature reaches 60°C
- Once the heat source is removed from the skin, the tissue temperature rapidly returns to normal, but the tissue injury does not stop at that point.

- Within the seconds of injury → arterioles spasm → red blood cells aggregation; Histologic changes are visible within 15 minutes, and tissue edema begins.

- Tissue edema typically peaks 6 to 12 hours postinjury.
- It is much easier for a burn injury to penetrate through the dermal layers in thin skin than in thick skin, leading to more severe injury in the former with a similar degree of heat transfer.
- These injuries pose a particular risk in infants and children as well as the elderly.
- Shortly after the burning stops, processes of both inflammation and healing begin.

Clinical Manifestations

Descriptions of burns are based on the depth of the injury and the extent of coverage

<u>Superficial burns</u>

- Involve the **epidermis alone.**
- These injuries are typically **painful and erythematous**, and they will **heal without scarring**.

These injuries are not included in calculations of the percentage total body surface area (TBSA) burned

<u>Superficial partial thickness burns</u>

- Involve the **entire epidermis and extend down to the papillary region of the upper dermis**.
- These injuries are **painful, erythematous, and moist**, and they form blisters.

<u>Deep partial thickness burns</u>

- Involve the **entire epidermis and extend deeper into the lower reticular dermis.**
- These wounds are typically painful and whitish
- May have less pain depending on the extent of damage to nerve endings in the skin.

<u>Full-thickness burns</u>

- Extend through the **epidermis and entire dermis and destroy nerves and other deep structures.**
- These wounds are **insensate, stiff, white or tan in color**

Wounds of this depth cannot heal spontaneously and will have significant scarring and contracture if not surgically managed.

TABLE 1 Types of Burns

DEPTH	ANATOMY	APPEARANCE	SENSATION	TIME	TREATMENT
Superficial	Epidermis only	Pink or red and dry, blanches	Painful	Days	Topical
Superficial partial thickness	Epidermis and upper papillary dermis	Pink, clear blisters, moist surface, blanches	Painful	14–21 days	Debride and topical medication
Deep partial thickness	Epidermis, papillary dermis, and lower reticular dermis	Pink or red, moist surface, blisters, may be hemorrhagic, does not blanch	Painful, may have decreased sensation	Weeks or may progress to full thickness	Debride, topical medication, possible surgery
Full thickness	Epidermis, dermis, and into underlying tissue	White or brown, dry, leathery, does not blanch	Insensate	Weeks, and heals by contraction and scarring	Surgical

<table>
<tr><th></th><th></th><th colspan="2">Partial-thickness (2nd degree)</th><th colspan="2">Full-thickness</th></tr>
<tr><th></th><th>Superficial (1st degree)</th><th>Superficial partial-thickness</th><th>Deep partial-thickness</th><th>Full-thickness (3rd degree)</th><th>Deep full-thickness (4th degree)</th></tr>
<tr><td>Depth</td><td>• Involves only the epidermis</td><td>• Involves the epidermis and the upper dermis</td><td>• Involves the epidermis, the upper dermis, and part of the deep dermis</td><td>• Involves the epidermis, the entire dermis, and causes damage to subcutaneous adipose tissues</td><td>• Involves the epidermis, entire dermis, subcutaneous adipose tissues, and underlying structures such as muscle, tendon, ligament, and/or bone</td></tr>
<tr><td>Clinical features</td><td>• Painful
• Often followed by desquamation
• No significant blister formation</td><td colspan="2">• Often nonpainful, may be painful if superficial partial-thickness
• Erythematous and blanch with pressure
• Deep partial thickness burns may be pale in color
• Characterized by blister formation</td><td colspan="2">• Often nonpainful, often a perception of pressure
• Commonly white and charred in appearance with eschar formation
• Blister formation is not typical
• Exposure of deep structures such as tendons, muscle, or bone</td></tr>
<tr><td>Treatment</td><td>• Supportive
• Consider loose bandage for coverage
• Consider nonsteroidal anti-inflammatory drugs (NSAIDs) or acetaminophen for pain control
• Topical antibiotics not usually necessary</td><td colspan="2">• Topical antibiotics such as silver sulfadiazine
• Consider loose bandage for coverage
• Consider NSAIDs or acetaminophen for pain</td><td colspan="2">• Surgical debridement and coverage with skin graft is often necessary
• Topical antibiotics with monitoring for signs of infection
• Aggressive pain control may be necessary, including with opiates</td></tr>
</table>

https://www.researchgate.net/figure/Classification-of-Thermal-Burn-Injury-by-Depth-3-7_tbl1_356609646

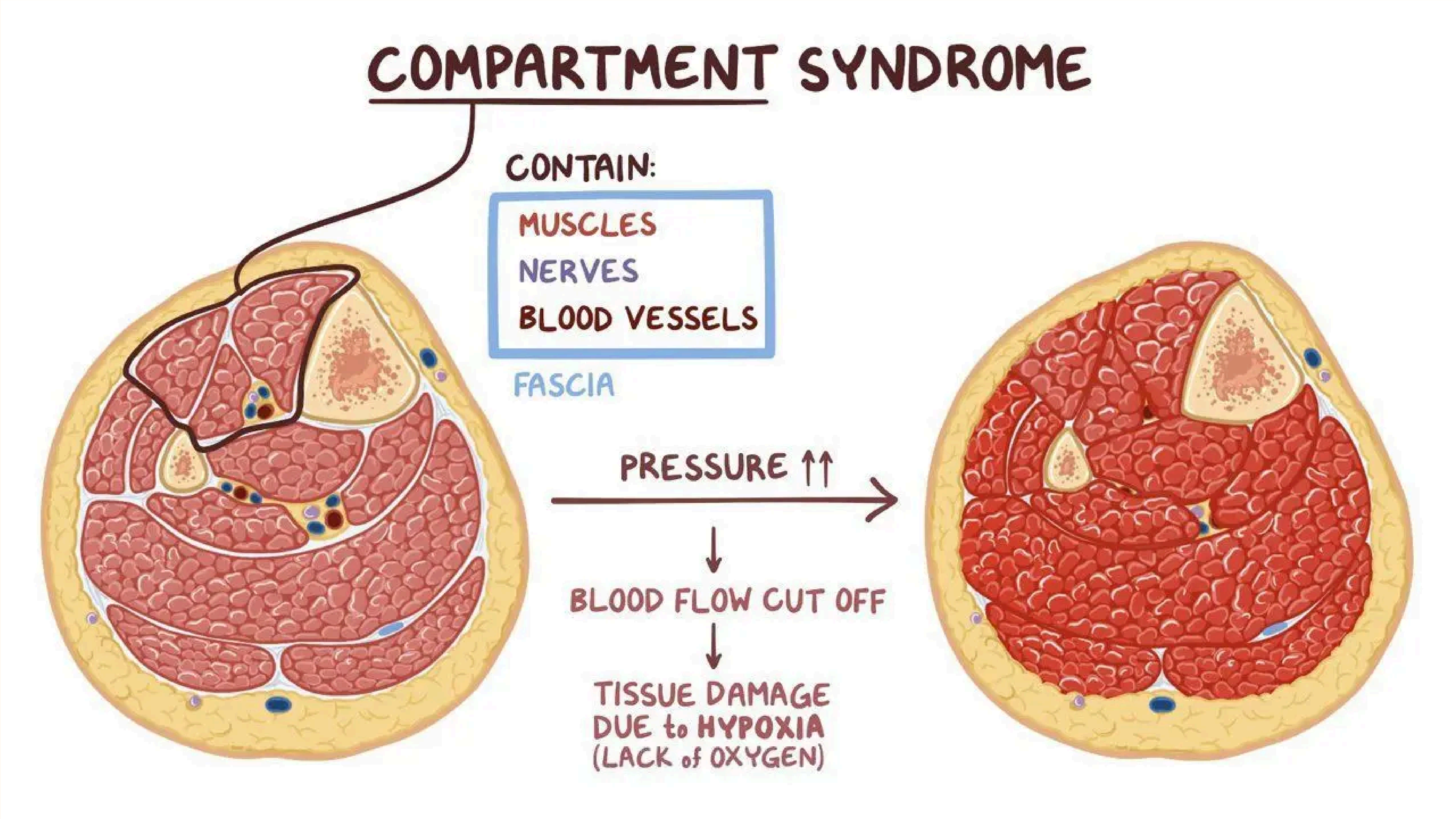
Complications

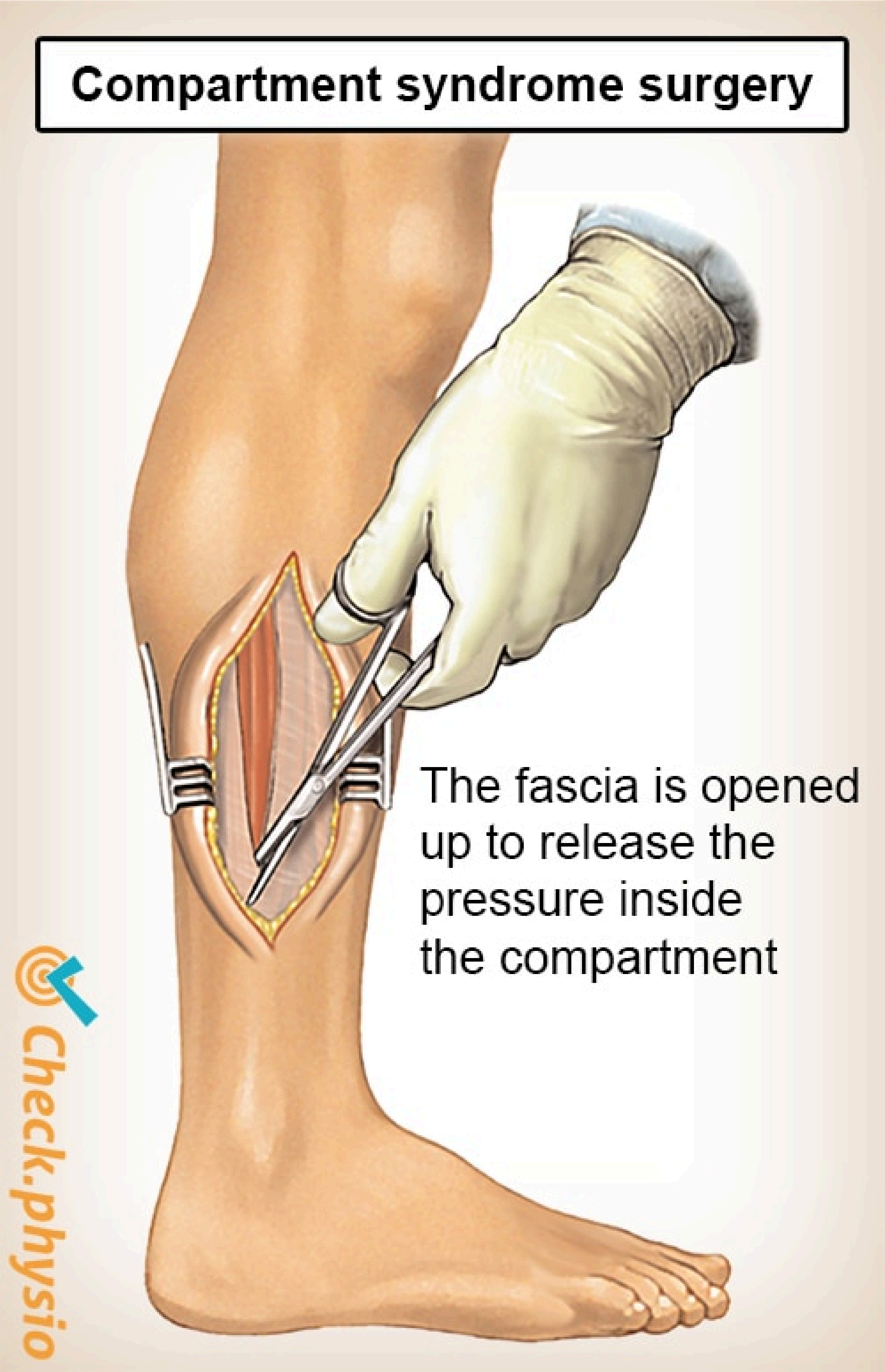
- Areas of burn that have extensive or circumferential deep partial thickness or full-thickness burns are at risk for development of restrictive eschars.

- Eschars can restrict circulation, leading to compartment syndrome in extremities and can decrease respiration if on the chest or neck.

- Ideally, burn patients can reach burn centers before these complications arise, but an escharotomy could be required urgently in some cases.

- Inhalation injury is a source of significant morbidity in burn patients.
- The initial injury may not seem particularly severe, but edema can develop rapidly in the airway, which makes delayed airway management perilous.
- Early intubation may be necessary.





https://www.osmosis.org/learn/Compartment_syndrome
https://www.physiocheck.co.uk/condition/101/compartment-syndrome-of-the-lower-leg

Special Situations

<u>Chemical Burns</u>

- The risk and degree of injury caused by chemical burns vary greatly, depending on the substance involved as well as on the concentration of the material and the duration of exposure.
- General precautions for chemical burns are to **<u>decontaminate as quickly as possible</u>** while avoiding rescuer contamination or injury.
- For dry or powdered chemicals, remove as much chemical as possible while it is still in the dry form by brushing or other mechanical removal.
- After mechanical removal, or for liquid contamination, **dilute the chemical and continue the removal process by using water**.
- Water should be used via copious, high-flow, low-pressure irrigation for at least 20 minutes.
- Evaluate the patient for possible inhalation or ocular injury as well.

TABLE 2 Chemical Burns*

CHEMICAL	TREATMENT	SPECIAL NOTES
Dry chemical	Brush or remove as much chemical as possible, then copious irrigation	Obtain material safety data sheet information on exposure
Liquid chemical	Copious irrigation	
Ocular injury	Copious irrigation with lactated Ringer's solution, measure pH early and repeat frequently	
Elemental metals (e.g., sodium, magnesium)	Cover with mineral oil if available, physically remove as soon as possible, irrigate with water if no other options	Water causes an intense exothermic reaction, but tissue damage occurs with continued contact
Strong alkalis (pH 10–12; e.g., lye, bleach, cement [calcium oxide], ammonia)	Copious irrigation, continue until pH normalizes but at least 1 hour; may require 12 or more hours	Current recommendations are not to try to neutralize, but research is continuing
Petroleum	Irrigation, mild soap solution	May be seen in vehicle trauma patients
Hydrofluoric acid	Irrigation, topical calcium gluconate (Calgonate)[1] mixed with water-based lubricant (e.g., K-Y jelly), intradermal or intravenous calcium gluconate[1]	Associated with pain out of proportion to appearance, fluoride ion can cause hypocalcemia and death
Phenol	Polyethylene glycol (PEG) (Miralax)[1] stops phenol burn more quickly than water; if PEG is not available, use high-flow, low-pressure, copious irrigation	

*Consider contacting a burn center or poison control center for assistance with chemical injury treatment.
[1]Not FDA approved for this indication.

Special Situations

Ocular Injuries

- Patients with any signs of burns to the face, **complaints of eye pain**, or inability to communicate should undergo for eye specialist consultation.

Electrical Injuries

- Injuries with electrical sources vary based on the type of circuit (alternating vs. direct current), amperage, voltage, tissue resistance, duration of exposure, and path of current through the body.
- High voltage (≥1000V) tends to cause more thermal tissue injury
- Depending on the path of electricity, significant internal injuries beneath normal-appearing skin
- Cutaneous burns due to electrical injury can be treated similarly to thermal burns.
- Assessment for other injuries:
  - EKG and cardiac monitoring, electrolytes, renal function studies, creatine kinase, troponin, and coagulation

Burn Management

- After evaluation and stabilization of the respiratory and circulatory status of the patient, and after stabilizing acute traumatic injuries (Primary survey)
- The next step is to **evaluate the extent of the burn injuries**.
- Removing the heat source and cooling the injured areas remain a priority.
- Cool water irrigation or saline-soaked gauze.
- Maintain normothermia.
- Estimate TBSA
- Fluid administration

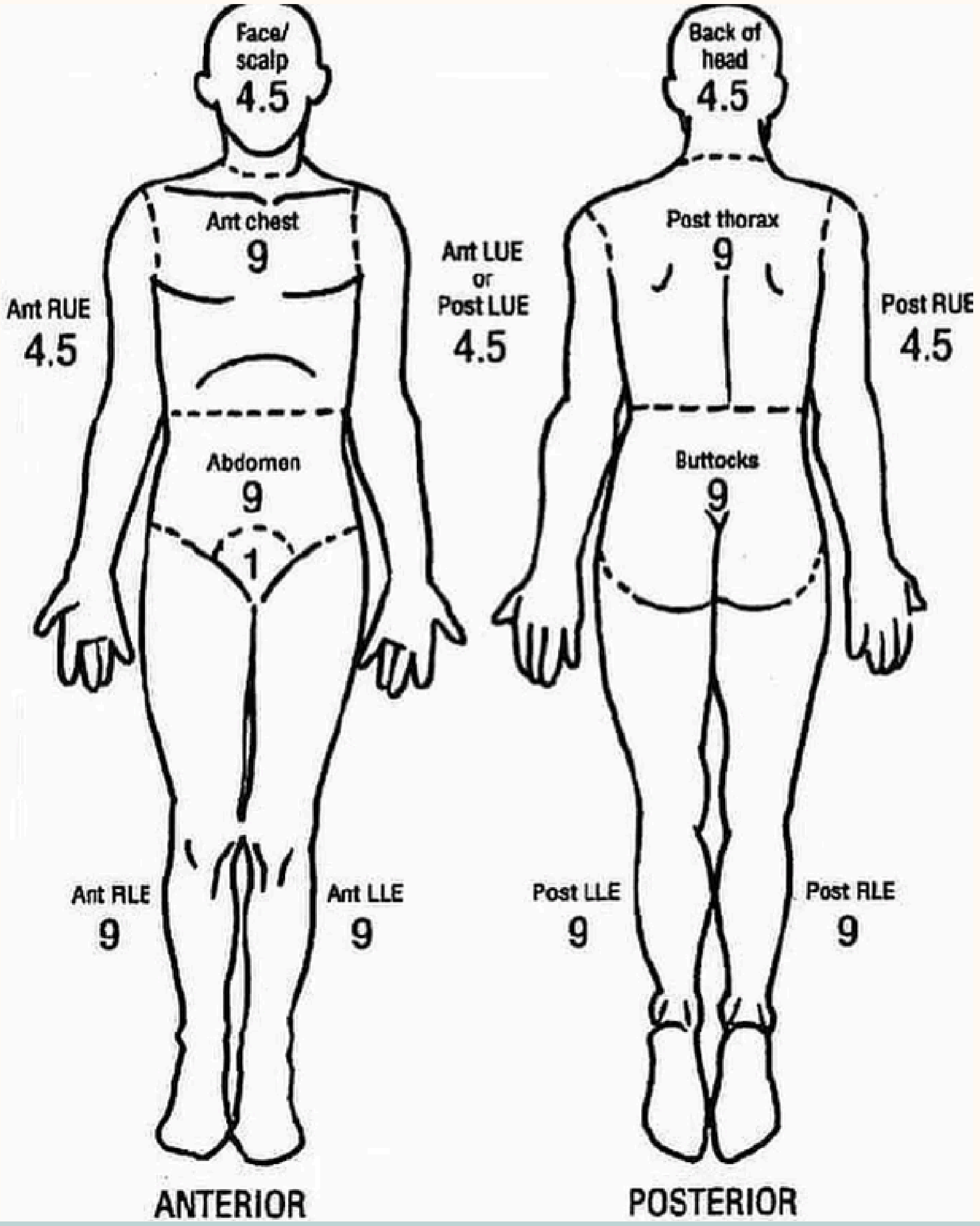
Estimate total body surface area (TBSA)

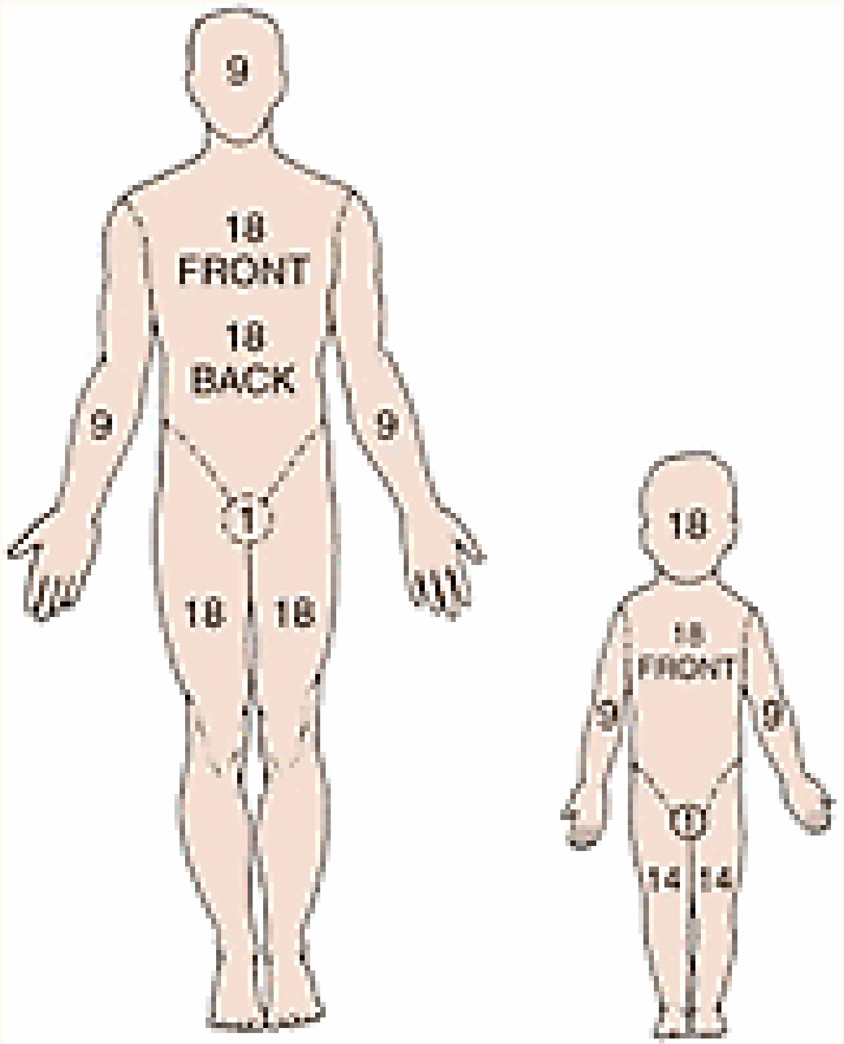
****

Superficial (first-degree) burns are <u>NOT</u> included in the percentage TBSA burn assessment.

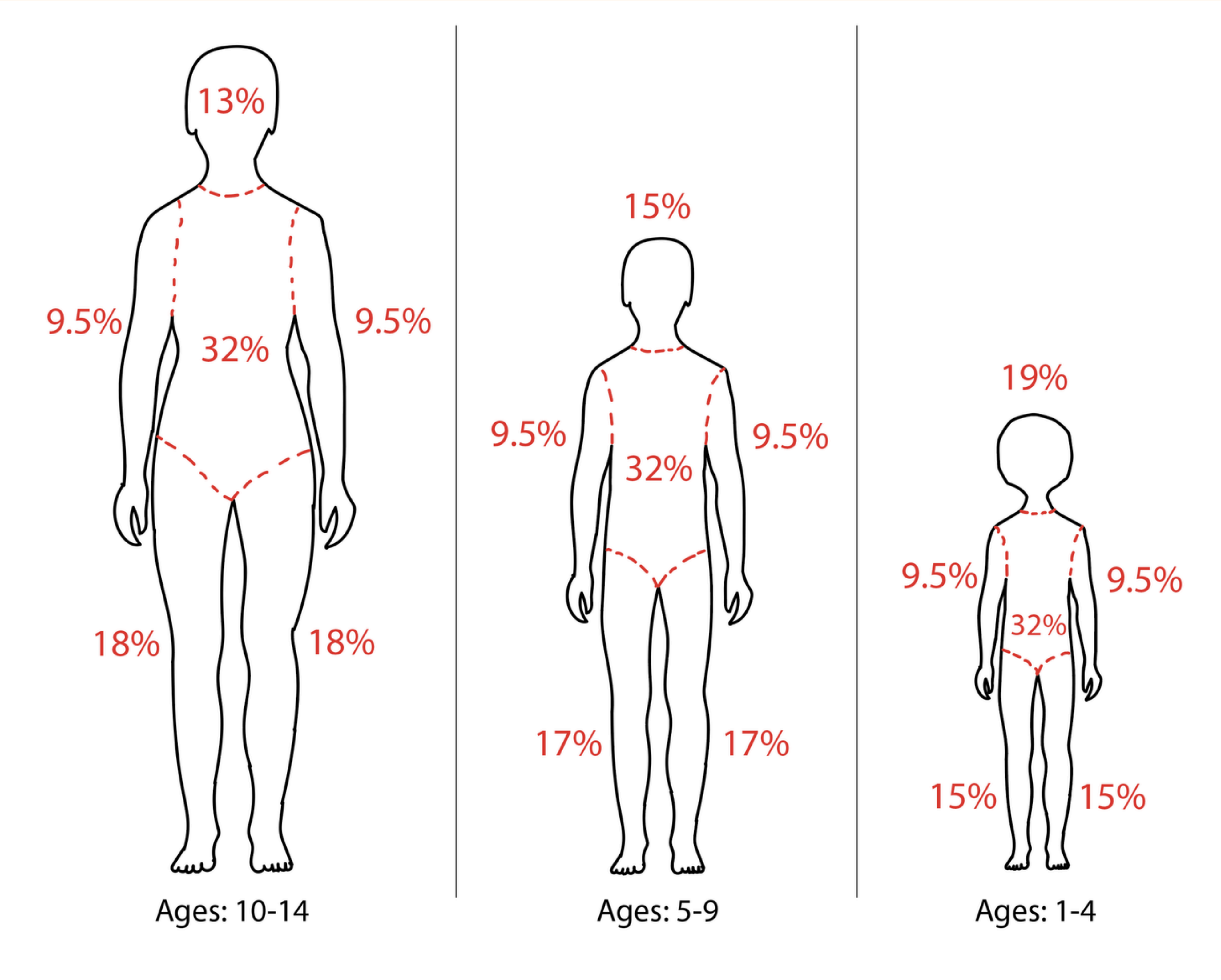
****

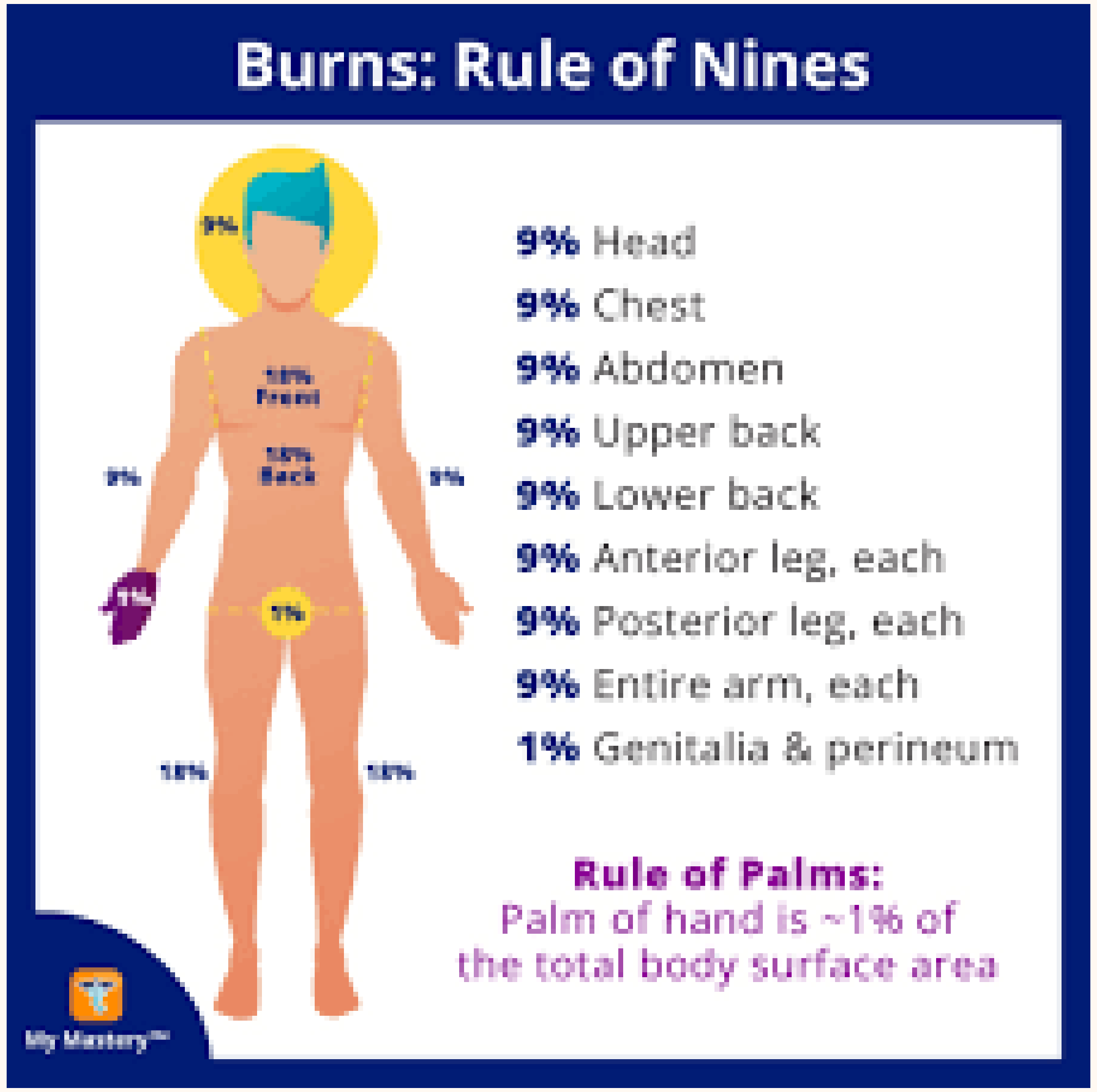
- The “rule of nines” diagram is an accepted mechanism for approximating the percentage of TBSA burned in adults, but it tends to overestimate.
- Because children have a proportionally larger head and torso, diagrams have been adapted for use in pediatric populations.
- The Lund-Browder chart can be utilized with patients of all ages





The entire head: 9% (4.5% for anterior and posterior)
The entire trunk: 36% (can be further broken down into 18% for the anterior torso and 18% for the posterior torso)
The anterior aspect of the trunk: Can further be divided into the chest and abdomen, each representing 9%
The upper extremities: 18% (includes 9% for each upper extremity)
Each upper extremity: Can be further divided into its respective anterior and posterior portions (4.5% each).
The lower extremities: 36% (18% for each); can be further divided into 9% for the anterior and 9% for the posterior aspect
The groin: 1%



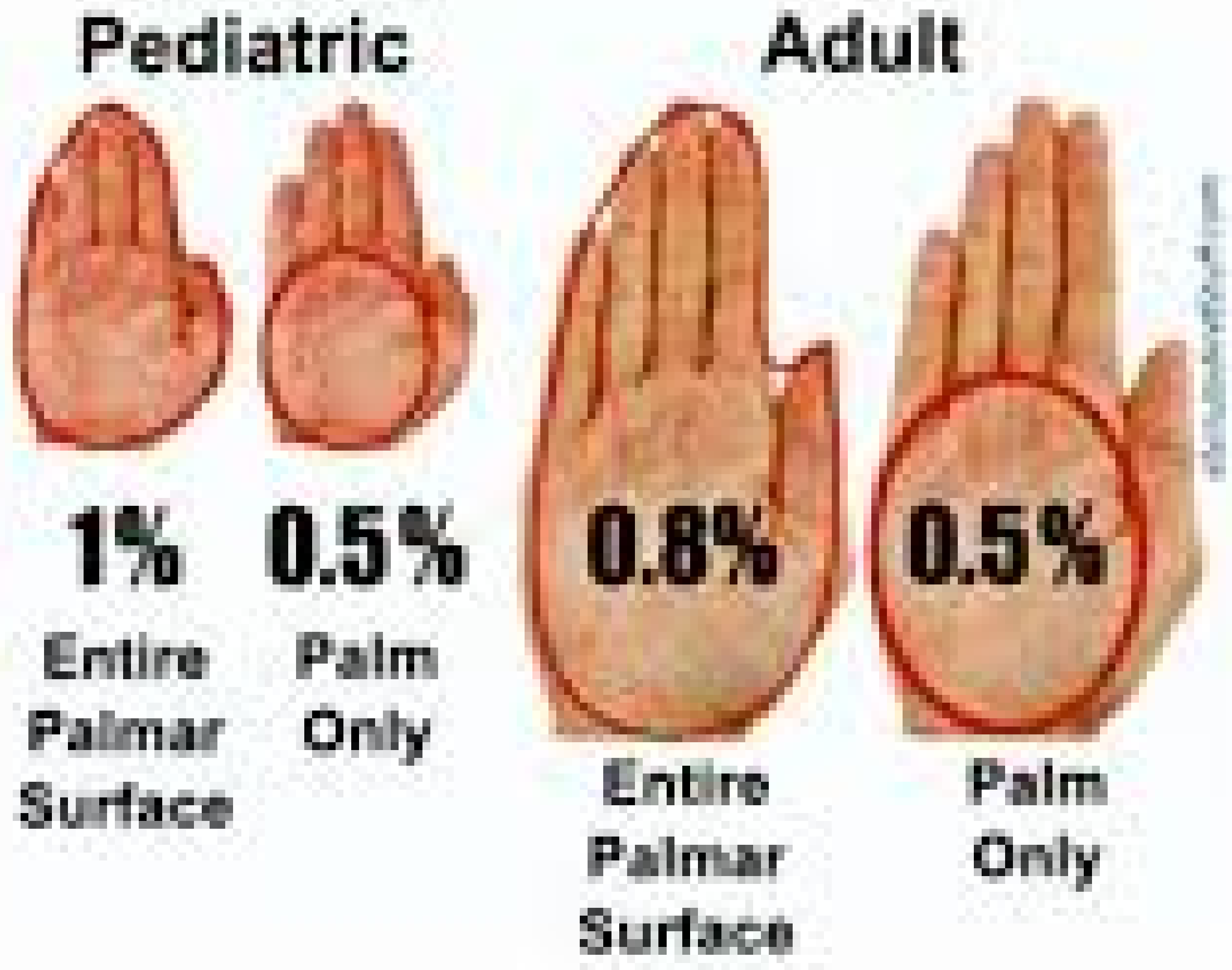


(Patient's palm)

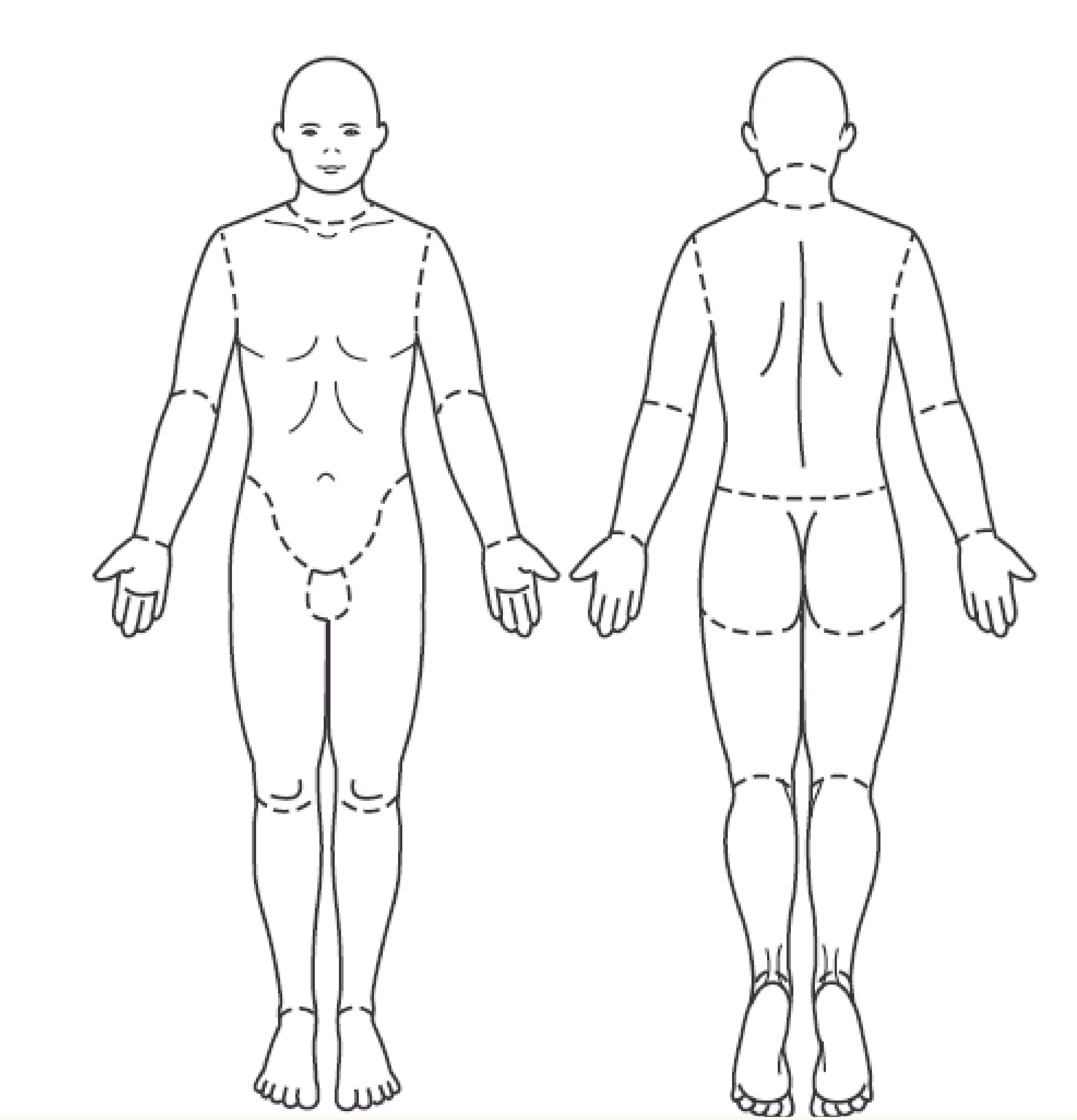
Rule of Palms

Use PATIENT's hand & ENTIRE palmar surface

Good for TBSA<15%



Lund-Browder classification of burn size



	Birth 1 yr.	1–4 yrs.	5–9 yrs.	10–14 yrs.	15 yrs.	Adult	Burn size estimate
Head	19	17	13	11	9	7	
Neck	2	2	2	2	2	2	
Anterior trunk	13	13	13	13	13	13	
Posterior trunk	13	13	13	13	13	13	
Right buttock	2.5	2.5	2.5	2.5	2.5	2.5	
Left buttock	2.5	2.5	2.5	2.5	2.5	2.5	
Genitalia	1	1	1	1	1	1	
Right upper arm	4	4	4	4	4	4	
Left upper arm	4	4	4	4	4	4	
Right lower arm	3	3	3	3	3	3	
Left lower arm	3	3	3	3	3	3	
Right hand	2.5	2.5	2.5	2.5	2.5	2.5	
Left hand	2.5	2.5	2.5	2.5	2.5	2.5	
Right thigh	5.5	6.5	8	8.5	9	9.5	
Left thigh	5.5	6.5	8	8.5	9	9.5	
Right leg	5	5	5.5	6	6.5	7	
Left leg	5	5	5.5	6	6.5	7	
Right foot	3.5	3.5	3.5	3.5	3.5	3.5	
Left foot	3.5	3.5	3.5	3.5	3.5	3.5	

Management of Inhalation Injury

BOX 1 Symptoms of Carbon Monoxide Exposure*

Headache
Dizziness
Nausea
Vomiting
Confusion
Fatigue
Chest pain
Shortness of breath
Loss of consciousness

*It is important to note that carboxyhemoglobin levels do not correlate well with symptoms or with severity of exposure.
Arterial blood gas samples are not superior to venous blood samples.

- On 100% oxygen as soon as possible
- Carbon monoxide binds tightly to hemoglobin, and by increasing the oxygen concentration in the lungs and blood, the half-life of carboxyhemoglobin is greatly reduced.
- The half-life of carboxyhemoglobin is 74 minutes.
- After 6 hours on 100% oxygen, if the patient is asymptomatic, treatment can be stopped.

Fluid Resuscitation and Monitoring

- The starting fluid requirements are 2 to 4 mL/kg of lactated Ringer's solution per TBSA percent value, with half given in the first 8 hours and the rest over the next 16 hours
- Urine output should be monitored hourly and maintained between 0.5 and 1 mL/kg/h
  - If urine output is greater than 1 mL/kg/h, the rate of fluid administration should be decreased by 20% and continued to be monitored hourly.
  - If the urine output is less than 0.5 mL/kg/h, the rate of fluid administration should be increased by 20% and continued to be monitored hourly.

BOX 2 Fluid Administration and Calculation

80-kg male patient with a burn of 35% TBSA full-thickness and partial thickness burns:

80 kg × 35 × 3 mL/kg = 8400 mL in first 24 hours

8400 mL/24 hours ÷ 2 = 4200 mL in first 8 hours, 4200 mL in next 16 hours

Initial rate of 525 mL/h over first 8 hours, then switching to a rate of 265 mL/h over next 16 hours

Expected urine output is 0.5–1 mL/kg/h

80 kg × 0.5 mL/kg/h = 40 mL/h

80 kg × 1 mL/kg/h = 80 mL/h

So, for our 80-kg patient, we would expect 40–80 mL of urine every hour. In hours 1 and 2 of resuscitation, the patient has 65 mL of urine per hour. In hour 3 of resuscitation, the patient has 90 mL of urine per hour. We would then decrease the fluid administration rate by 20%, or

525 mL/h–(0.20 × 525) = 420 mL/h

Thus, the rate of administration of lactated Ringer's solution would change from 525 mL/h to 420 mL/h and continue to be monitored and recorded hourly, with continued adjustments based on hourly urine output.

TBSA, Total body surface area.

The Parkland Formula

Apply only in 2nd and 3rd degree burns.

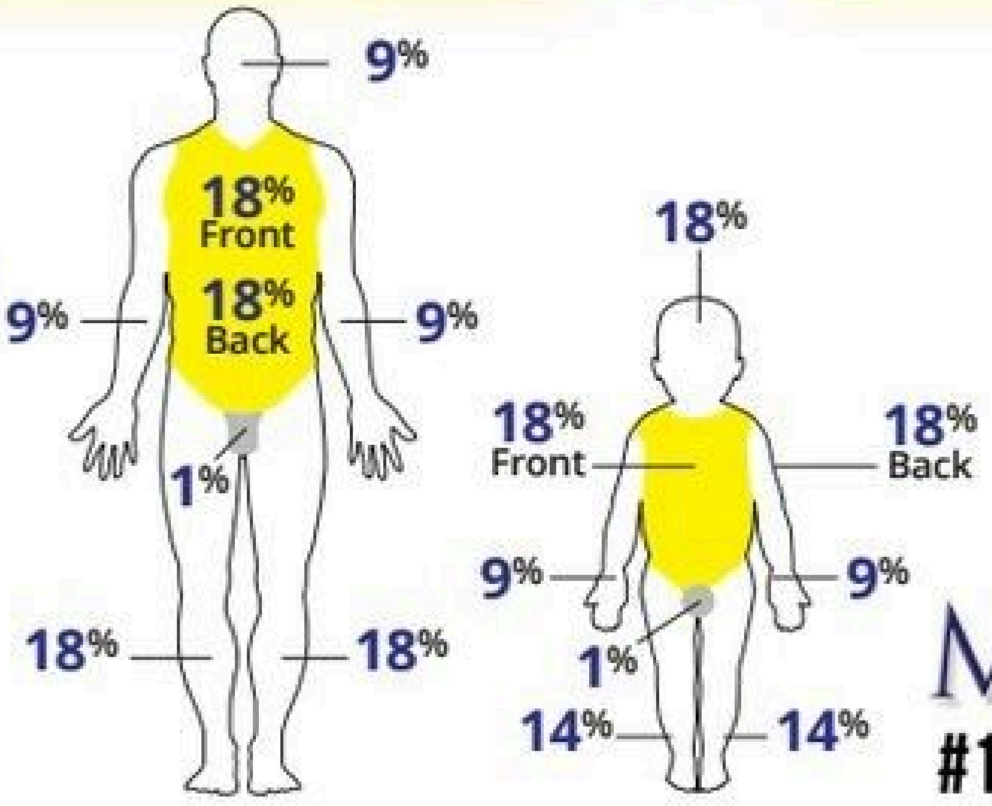
Volume of Lactated Ringers Solution

4 mL x BSA (%) **x Body Weight** (kg)

(Body Surface Area)

Give half of the solution for the **First 8 Hours**	Give half of the solution for the **First 16 Hours**

Rule of Nines



Patient's hand approximates **1%** total body surface area.

MEDIC TESTS

#1 EMT & PARAMEDIC EXAM PREP

mEq/L	Normosol-R and Plasma-Lyte A	Lactated Ringers	NaCl 0.9%	Human Plasma
Na	140	130	154	136 - 145
Cl	98	109	154	96 - 106
K	5	4	0	3.5 - 5.0
Mg	3	0	0	1.5 - 2.5
Ca	0	3	0	4.5 - 5.5 (ionized)
Lactate*	0	28	0	
Acetate*	27	0	0	
Gluconate*	23	0	0	
Osmolarity	294	273	308	275 - 295
pH	~7.4	6.0-7.5	4.5-7.0	7.35-7.45

แนวทางการรักษาพยาบาลผู้ป่วยทางศัลยกรรม
โรค : บาดแผลไฟไหม้ น้ำร้อนลวก (Burns)
โดย ราชวิทยาลัยศัลยแพทย์แห่งประเทศไทย

https://www.rcst.or.th/web-upload/filecenter/CPG/Burn.html

แนวทางในการรักษา วิธีการรักษาแตกต่างกันตามความรุนแรงของบาดแผลไฟไหม้ โดยอาศัยจากความลึก และขนาดของบาดแผลไฟไหม้ดังกล่าวแล้ว
โดยจำแนกแยกกลุ่มของคนไข้ออกเป็น 3 กลุ่ม

1. กลุ่มที่ได้รับบาดเจ็บไม่รุนแรง หรือรุนแรงน้อย สามารถให้การรักษาแบบคนไข้นอกได้
- First degree burn
- Second degree burn
  - ในเด็ก TBSA < 10%
  - ในผู้ใหญ่ TBSA <15%
- Third degree burn ที่ประเมิน TBSA < 2%

2. กลุ่มที่ได้รับบาดเจ็บรุนแรงมาก ต้องรับไว้ในโรงพยาบาล
- Second degree burn
  - ในเด็ก TBSA 10-15%
  - ในผู้ใหญ่ TBSA 15-30%
- Third degree burn ที่ TBSA 2-10%
- มีบาดแผลไฟไหม้ที่บริเวณ**ใบหน้า, มือ, เท้า, บริเวณ *perineum***
- มีบาดแผลเกิดจากไฟฟ้าช็อต, บาดแผลจากการสัมผัสกับสารเคมี, มี inhalation injury ร่วมด้วยหรือสงสัยว่าจะมี
- มีโรคทางอายุรกรรมร่วมด้วย หรือ มีกระดูกหักบริเวณที่มีบาดแผลไฟไหม้ หรือ มีการบาดเจ็บของอวัยวะหลายอย่างร่วมด้วย

3. กลุ่มที่ได้รับบาดเจ็บรุนแรงในระดับอันตราย ควรรับไว้รักษาในโรงพยาบาลที่มีศูนย์ดูแลรักษาคนไข้ไฟไหม้น้ำร้อนลวก (Burn Center) โดยเฉพาะ
- Second degree burn
  - ในเด็ก TBSA > 15%
  - ในผู้ใหญ่ TBSA > 30%
- Third degree burn ที่ประเมิน TBSA > 10%

Wound healing

Healing of Skin Wounds

Based on the nature and size of the wound, the healing of skin wounds is said to occur by first or second intention.

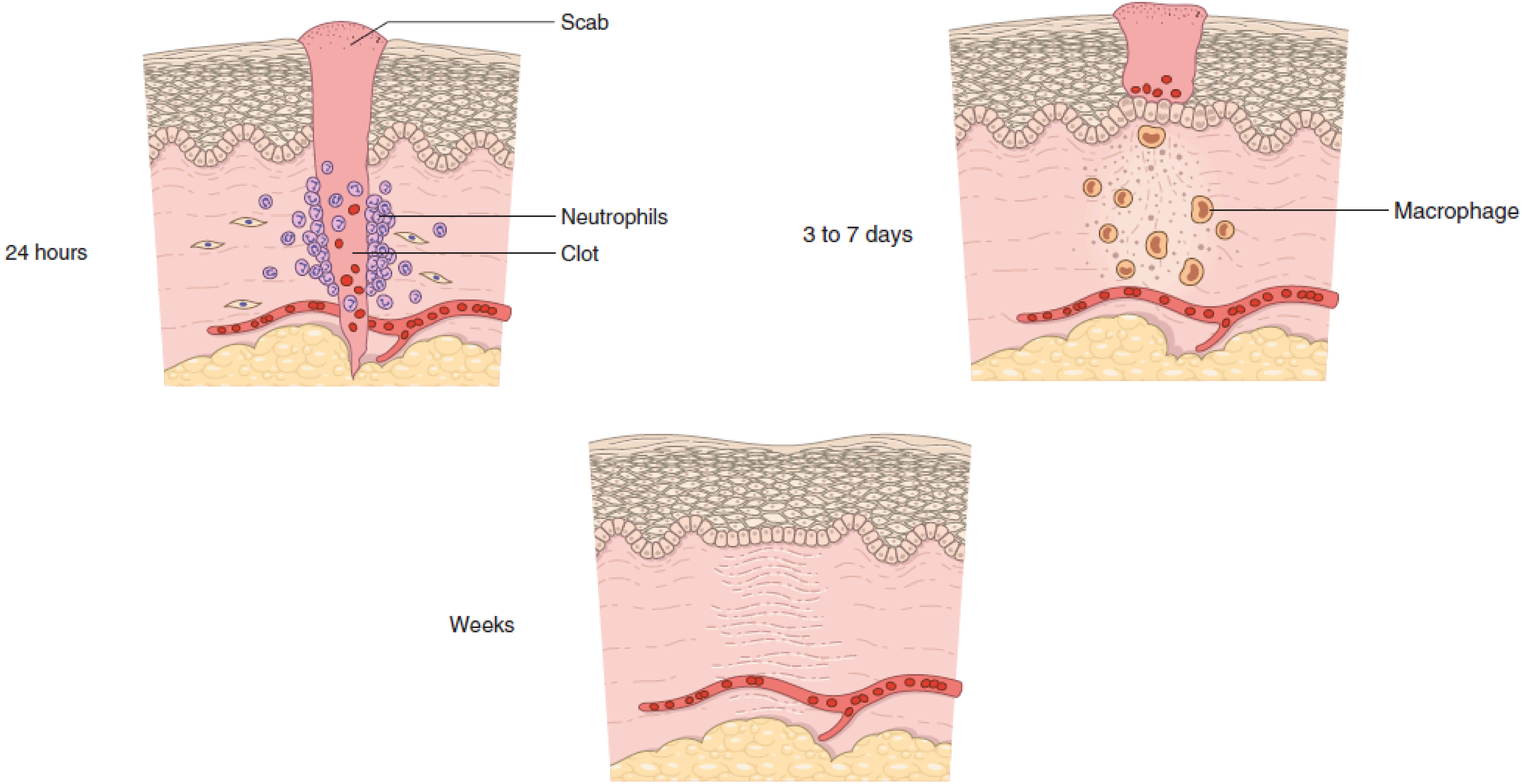
Healing by First Intention

- Involves only the epithelial layer
- Epithelial regeneration (primary union or healing by first intention)
- Example: healing of a clean, uninfected surgical incision approximated by surgical sutures.

Wounding → activation of coagulation pathways → formation of a blood clot on the wound → clot stop bleeding and supports migrating cells

- Within 24 hours: neutrophils migrating
- Within 24 to 48 hours: epithelial cells from both edges have begun to migrate and proliferate
- By day 3: neutrophils replaced by macrophages, and granulation tissue
- By day 5: neovascularization reaches its peak, new edematous granulation tissue
- The second week: continued collagen accumulation and fibroblast proliferation
- The end of the first month, the scar comprises a cellular connective tissue, devoid of inflammatory cells and covered by an essentially normal epidermis

HEALING BY FIRST INTENTION



Healing of Skin Wounds

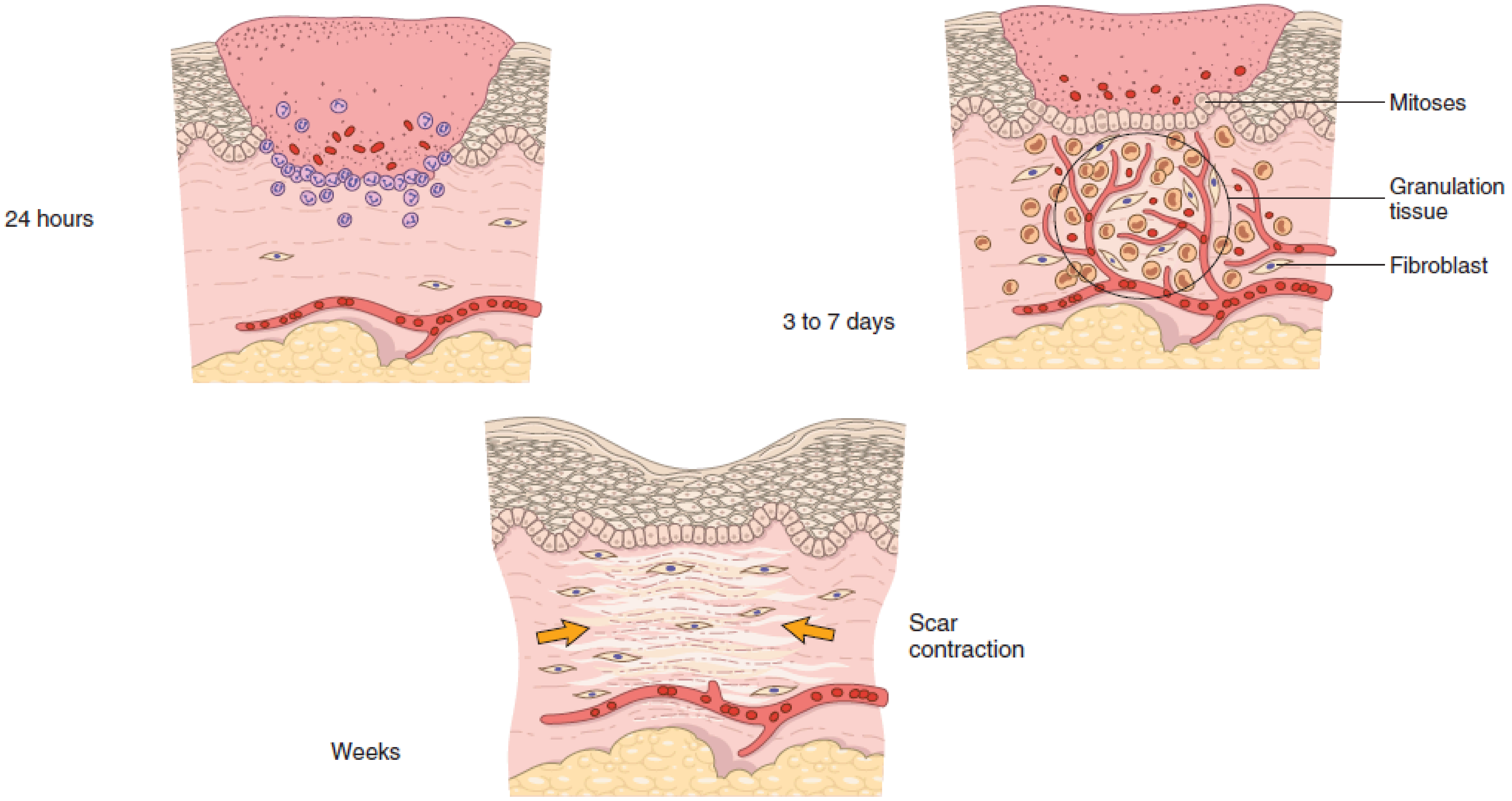
Healing by Second Intention (secondary union)

- In large wounds causing tissue deficits, the fibrin clot is larger, more exudate and necrotic debris
- Inflammation is more intense (to remove necrotic debris, exudate, and fibrin)
- Larger granulation tissue are formed
- A greater volume of granulation tissue results in a greater mass of scar tissue

- At first a provisional matrix containing fibrin, plasma fibronectin, and type III collagen is formed
- But in about 2 weeks this is replaced by a matrix composed primarily of type I collagen

- The original granulation tissue is converted into avascular scar.
- The dermal appendages that have been destroyed are permanently lost
- By the end of the first month, the scar is made up of acellular connective tissue, covered by intact epidermis.
- Wound contraction helps to close the wound by decreasing the gap between its dermal edges and by reducing the wound surface area (an important feature in healing by secondary union)
- Within 6 weeks, large skin defects may be reduced to 5% to 10% of their original size, largely by contraction.

HEALING BY SECOND INTENTION



Factors That Influence Tissue Repair

Infection: causes of delayed healing; it prolongs inflammation and potentially increases the local tissue injury.

Diabetes: compromises tissue repair and is one of the most important systemic causes of abnormal wound healing.

Nutritional status: protein deficiency and vitamin C deficiency inhibit collagen synthesis and retard healing.

Glucocorticoids (steroids): anti-inflammatory effects

Factors That Influence Tissue Repair

Mechanical factors: increased local pressure or torsion may cause wounds to pull apart, or dehisce.

Poor perfusion: arteriosclerosis, and diabetes or obstructed venous drainage (e.g., in varicose veins), also impairs healing.

Foreign bodies: such as fragments of steel, glass, or even bone impede healing by perpetuating chronic inflammation.

The type and extent of tissue injury and the character of the tissue

- Complete restoration can occur in tissues composed of stable and labile cells
- Permanent cells results in scarring and some loss of function

The location of the injury is also important

Abnormalities in Tissue Repair

Defects in Healing: Chronic Wounds

Venous leg ulcers

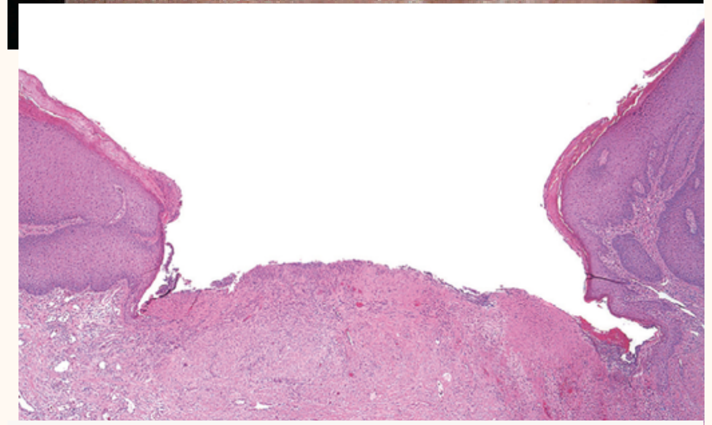
- A result of chronic venous hypertension (severe varicose veins or congestive heart failure)
- Deposits hemosiderin
- These ulcers fail to heal because of poor delivery of oxygen to the site of the ulcer

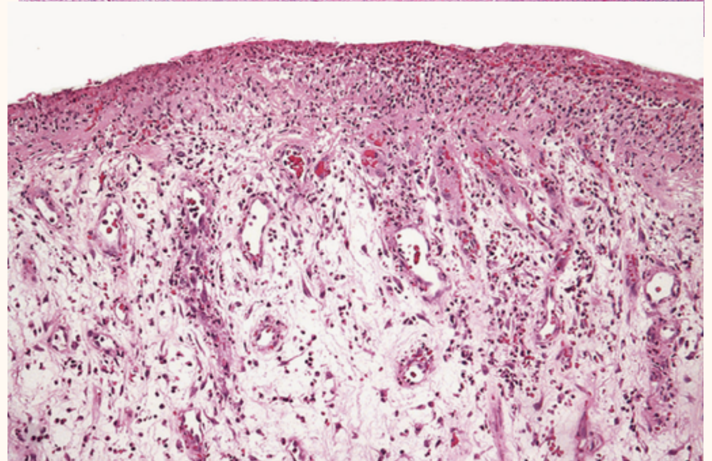
Arterial ulcers

- Atherosclerosis of peripheral arteries, especially associated with diabetes
- The ischemia results in atrophy and necrosis of the skin and underlying tissues
- These lesions can be quite painful.

Abnormalities in Tissue Repair

Defects in Healing: Chronic Wounds



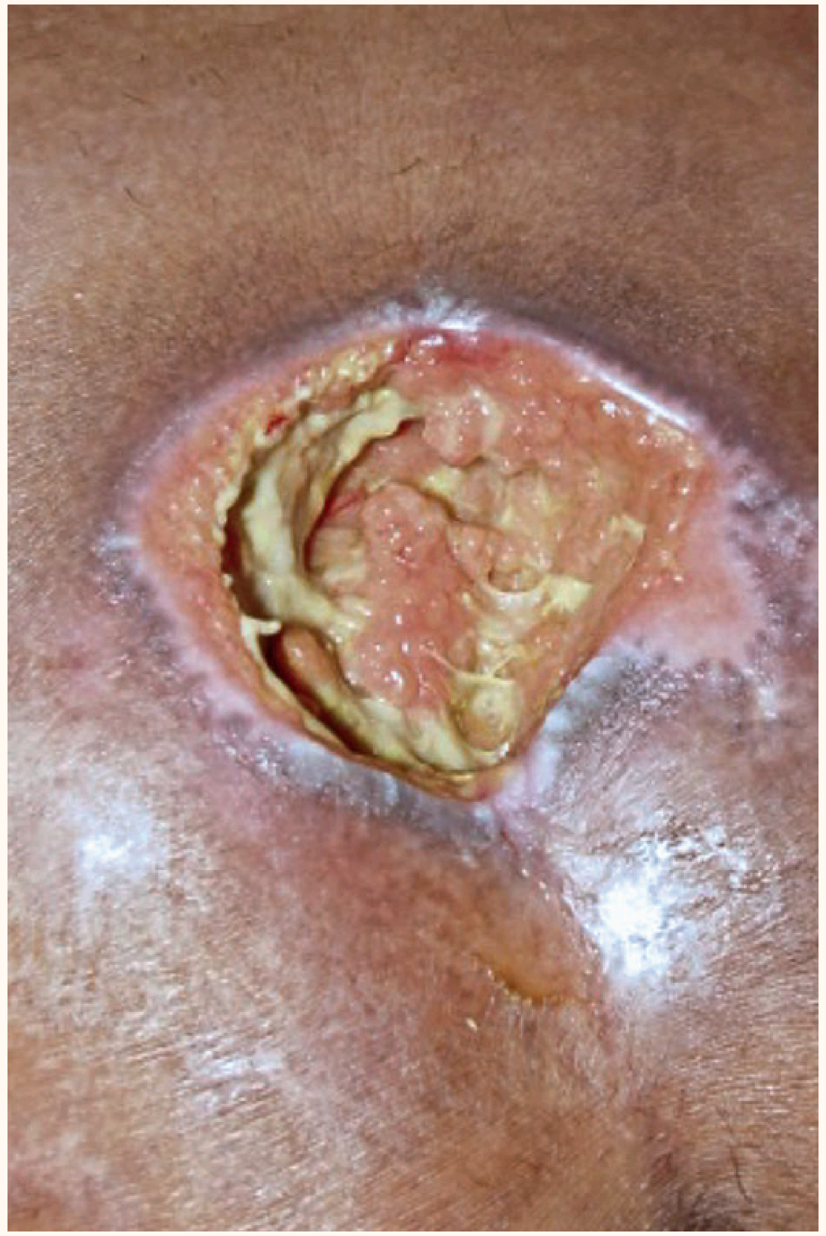


Diabetic ulcers

- Affect the lower extremities, particularly the feet
- There is tissue necrosis and failure to heal as a result of vascular disease causing ischemia, neuropathy, systemic metabolic abnormalities, and secondary infections
- Histologically, these lesions are characterized by epithelial ulceration and extensive granulation tissue in the underlying dermis

Pressure sores

- Areas of skin ulceration and necrosis of underlying tissues caused by prolonged compression of tissues against a bone
- The lesions are caused by mechanical pressure and local ischemia
- The risk factors for such an occurrence are obesity, malnutrition, infections, and vascular insufficiency



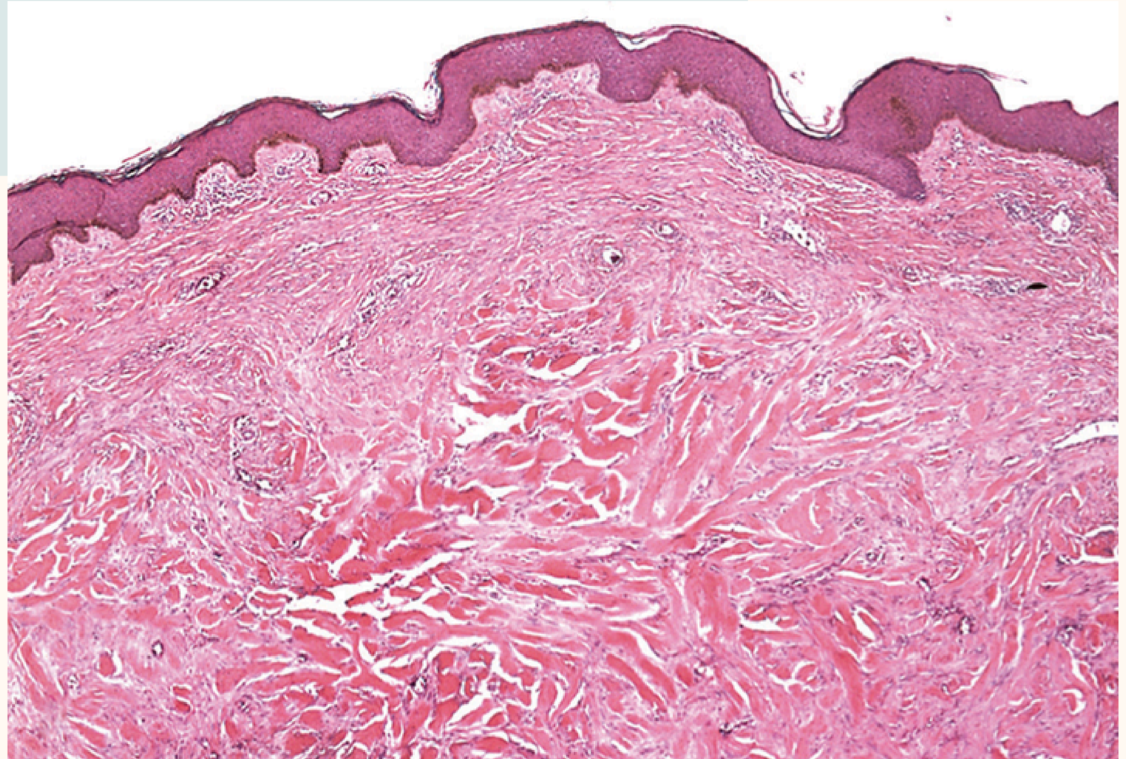
Abnormalities in Tissue Repair

Excessive Scarring

Excessive formation of the components of the repair process can give rise to hypertrophic scars and keloids

The accumulation of excessive amounts of collagen may give rise to a raised scar known as a hypertrophic scar
These often grow rapidly and contain abundant myofibroblasts, but they tend to regress over several months

If the scar tissue grows beyond the boundaries of the original wound and does not regress, it is called a keloid



References

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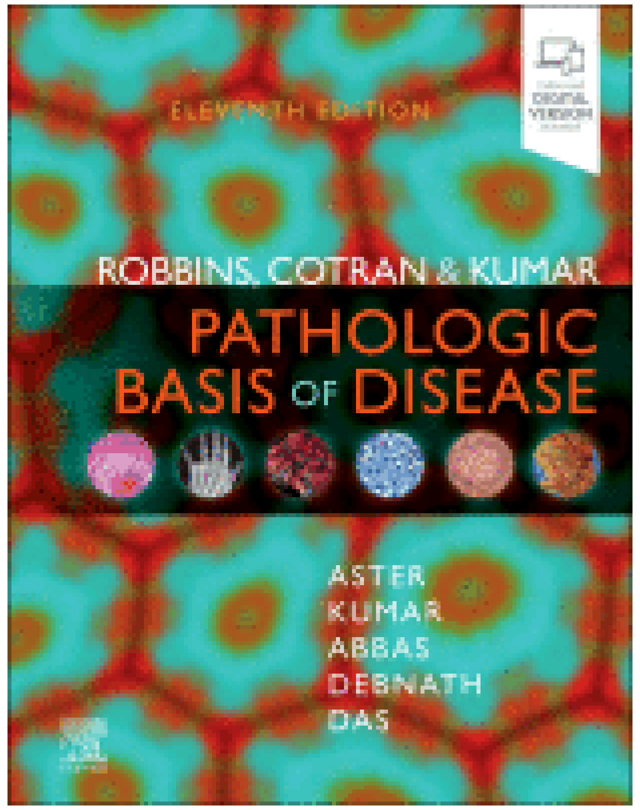

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Thank you

B4.2.3 Traumatic and mechanical disorders

กลุ่มที่ 1 และ 2	กลุ่มที่ 3
(1) Burns	(1) Keloid, hypertrophic scar
(3) Scar	
(4) Ulcers	
(5) Wound	