Pathophysiology-Local response

- Zone of coagulation
- Zone of stasis
- Zone of hyperemia

Local Response

- Zone of coagulation
  - Point of maximal damage
  - Irreversible tissue loss

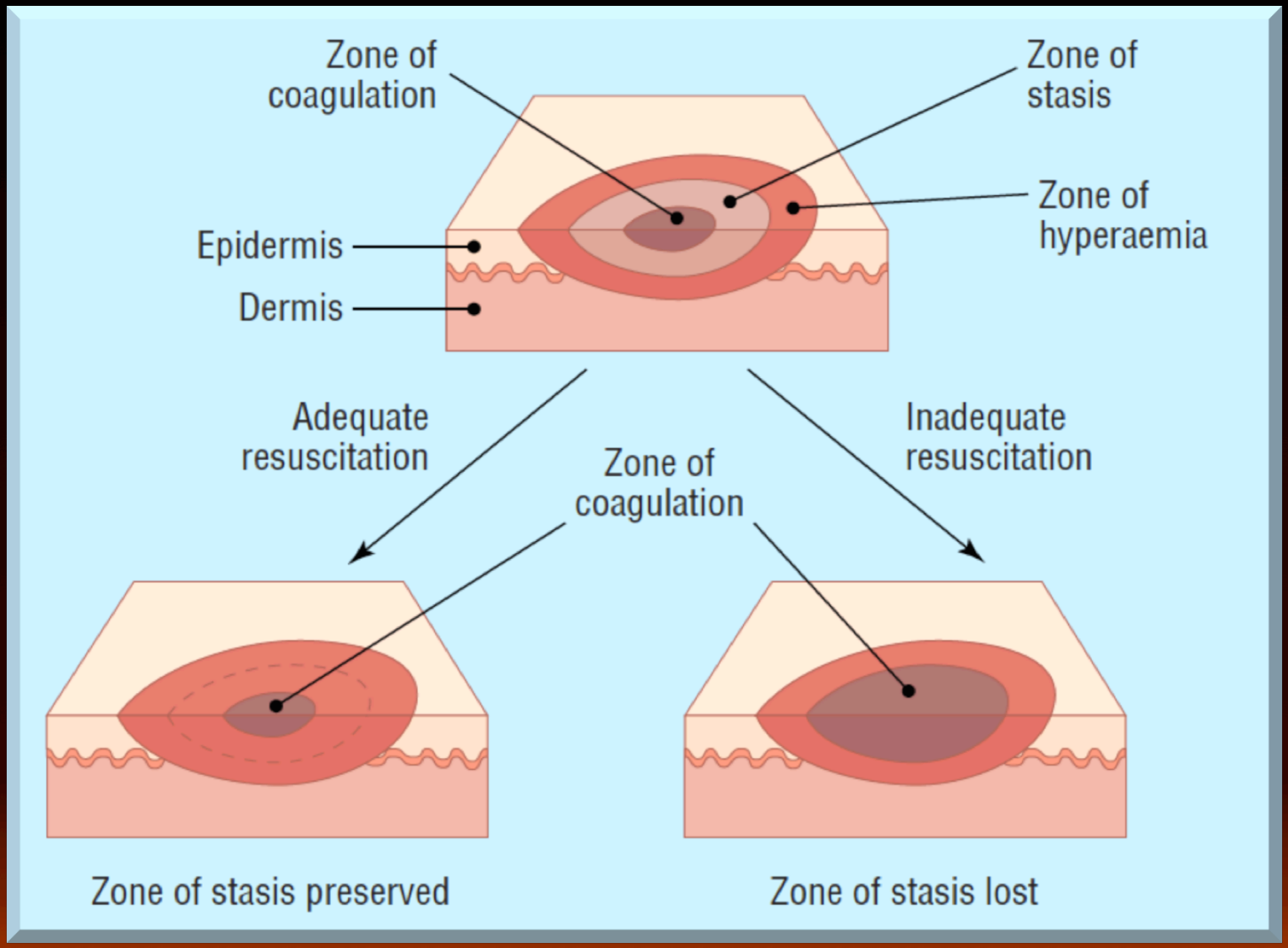
Local Response

- Zone of coagulation
- Zone of stasis
  - Decreased tissue perfusion
  - Potentially salvegable

Local Response

- Zone of coagulation
- Zone of stasis
- Zone of hyperemia
  - Invariably recovers unless prolonged hypotension

Local Response



Systemic response

- Represented as 2 phases
  - Resuscitation phase
  - Hyperdynamic, metabolic phase

Resuscitation phase

- Starts at time of injury
- Lasts 24-72 hours
- Characterized by
  - Intravascular volume depletion
  - Increased vascular permeability
  - Edema formation
  - Reduced cardiac output

Resuscitation phase

- Mechanisms driving intravascular volume depletion
  - Increased capillary permeability→loss of intravascular protein and fluids to intersitium
  - Disruption of sodium-ATPase pump→increased intracellular sodium concentration

Resuscitation phase

- Inflammatory mediators of edema and systemic derangements
  - ROS
    - Overproduction of ROS overwhelming the natural antioxidant capabilities
    - Inflammatory cells accumulate within the tissues Contributes to organ damage in severe burn injuries

Resuscitation phase

- Inflammatory mediators of edema and systemic derangements
  - ROS
  - Histamine
    - Released from mast cells w/in minutes of tissue damage
    - Contraction of the venular endothelial cells→ intracellular gaps
      - Increases vascular permeability
    - Increased hydrostatic pressure exacerbates burn associated edema

Resuscitation phase

- Inflammatory mediators of edema and systemic derangements
  - ROS
  - Histamine
  - Prostaglandins/prostacyclins/thromboxanes
    - Released from burned tissues
    - Mediate fever and pain
    - Prostaglandin/prostacyclin cause vasodilation→edema from altered intravascular hydrostatic pressures
    - Thromboxane causes vasoconstriction

Resuscitation phase

- Inflammatory mediators of edema and systemic derangements
  - ROS
  - Histamine
  - Prostaglandins/prostacyclins/thromboxanes
  - Kinins (bradykinin)
    - Produced at burn injury site
    - Causes venular dilation and increased microvascular permeability, smooth muscle contraction, and pain

Resuscitation phase

- Mechanisms of reduced cardiac output:
  - Reduced plasma volume
    - As discussed before
    - Results in a reduced preload

Resuscitation phase

- Mechanisms of reduced cardiac output:
  - Reduced plasma volume
  - Increased afterload
    - Driven by a sympathetic stimulation and hypovolemia
    - Mediated by release of catecholamines, vasopressin, angiotensin II and thromboxane A2-potent vasoconstrictors

Resuscitation phase

- Mechanisms of reduced cardiac output:
  - Reduced plasma volume
  - Increased afterload
  - Reduced myocardial contractility
    - Due to circulating myocardial depressant factors
    - Likely multifactorial
    - TNF-alpha results in release of including Il-6 and Il-1B
    - Cardiomyocyte apoptosis driven by caspase activation
    - May be associated with macrophage migration inhibitor factor

Resusciation phase

- Goals:
  - Restore and preserve tissue perfusion
  - Avoid ischemia and cellular shock
  - Avoid or support other organ dysfunctions

Hyperdynamic phase

- Characterized by:
  - Decreased peripheral vascular resistance
  - Increased cardiac output
  - Marked increase in metabolic rate

Hyperdynamic phase

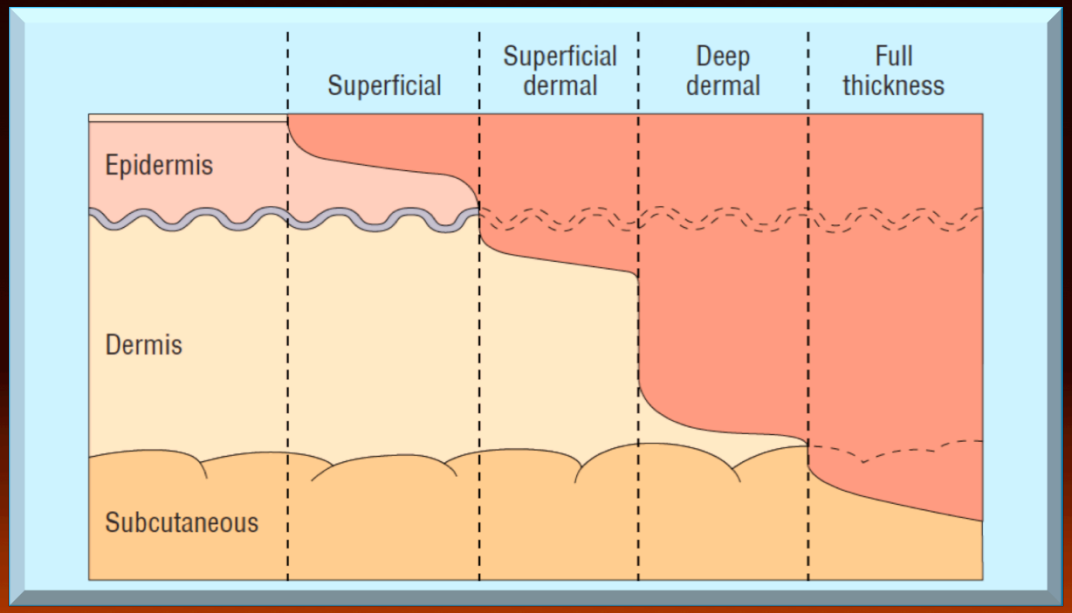
- Pathophysiology of hypermetabolic state
  - Protein catabolism
  - Gluconeogenesis
  - Glycogenolysis
  - Lipolysis
  - Hepatic insulin resistance
  - Increased glucose and oxygen consumption
  - Decreased lean body mass
  - Fever

Hyperdynamic phase

- Pathophysiology of hypermetabolic state
  - Mediated by augmented release of counter regulatory hormones
    - Cortisol
    - Glucagon
    - Catecholamines

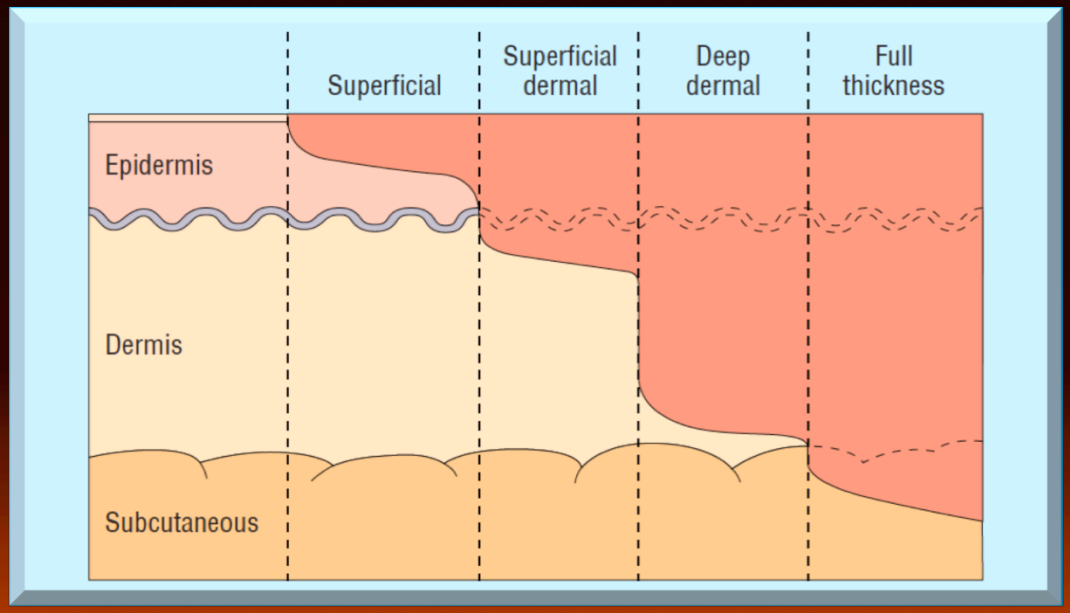
Burn Depth

- Partial thickness
  - Superficial: only epidermis, no dermis
  - Superficial dermal: through epidermis and upper layers of the dermis, through the papillary layer



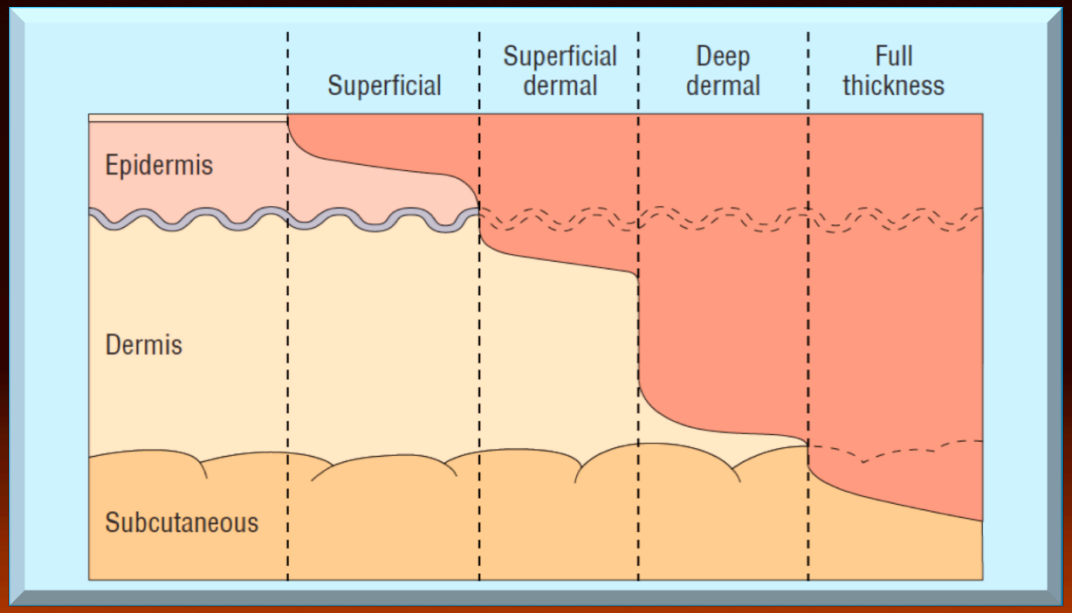
Burn Depth

- Partial thickness
- Deep dermal: through epidermis and into deep layers of the dermis, not through all of the dermis



Burn Depth

- Partial thickness
- Deep dermal
- Full thickness: through all skin layers into hypodermis



Burn Depth

Classification of burn wound	Dermal layers involved	Wound characteristics	Healing
Superficial	Epidermis only	Erythematous desquamation Dry, flaky appearance	Heals in 3–5 days via re-epitheliazation Minimal scar formation
Superficial partial-thickness	Epidermis	Erythematous, moist blanches	Heals in 1–2 weeks via re-epithelialization
	Upper 1/3 of dermis (papillary layer)	Painful blisters may be present Edema may be present Eschar formation	Minimal scar formation
Deep partial-thickness	Epidermis All dermis	Red-waxy white Reduced pain sensation Blisters absent Eschar formation	Heals in 2–3 weeks Recommend surgical intervention to prevent significant scar formation
Full-thickness	Epidermis dermis Subcutaneous tissue	Bloodless pearl-white Eschar formation Hair easily plucked	Requires surgical intervention

Estimating Burn Depth

- Bleeding test
  - Prick with 21 gauge needle
  - Superficial or superficial dermal: Brisk bleeding
  - Deep dermal: Delayed bleeding
  - Full thickness: No bleeding

Estimating Burn Depth

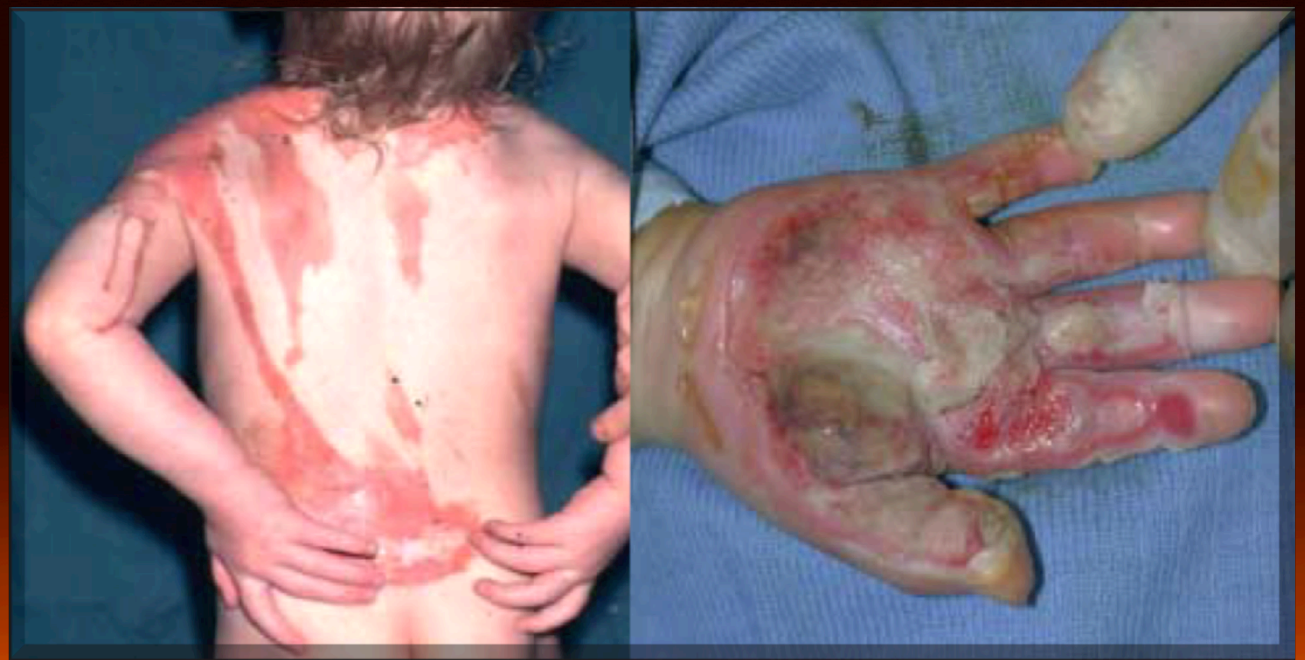
- Bleeding test
- Sensation
  - Superficial or superficial dermal: obviously painful
  - Deep dermal: Non-painful but retained sensation
  - Full thickness: Insensate

Estimating Burn Depth

- Bleeding test
- Sensation
- Appearance and blanching to pressure
  - Superficial: Red, moist wound, obvious blanching, rapid refill
  - Superficial dermal: Pale, dry but blanching wound that slowly regains colour
  - Deep Dermal: Mottled cherry red, no blanching
  - Full thickness: Dry, leathery, hard wound that does not blanch

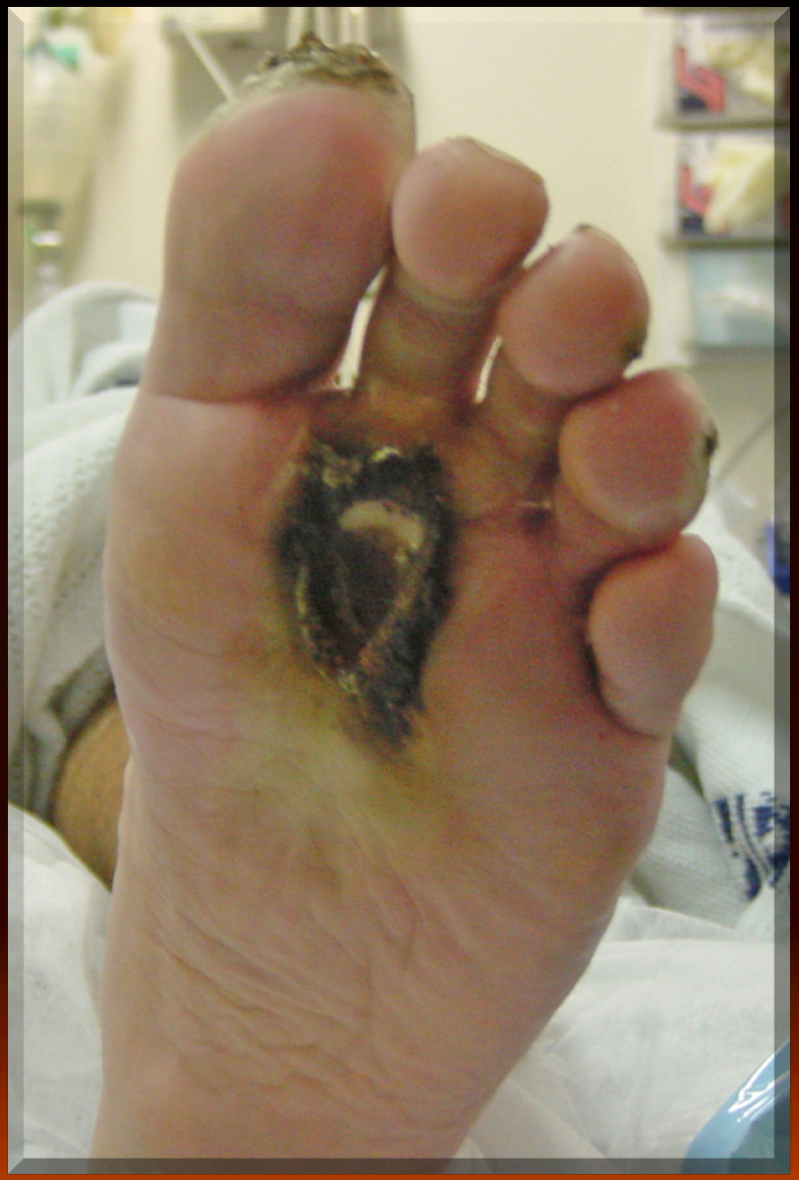
Mechanisms of injury

- Thermal injuries
  - Scald-superficial to superficial dermal burns
  - Flame-deep dermal or full thickness burns
  - Contact-deep dermal or full thickness burns



Mechanisms of injury

- Thermal injuries
- Electrical injury
  - Current travels through the body-creates and entry and exit point
    - Tissue between points can be damaged by current
    - Amount of heat generated=0.24 x $(\text{voltage})^2$ x resistance
    - Divided into two categories



Mechanisms of injury

- Thermal injuries
- Electrical injury
  - Low voltage
    - Typically due to exposure to domestic electrics
    - Causes small, deep contact burns at entry and exit site
    - Alternating nature of domestic currents→arrhythmias

Mechanisms of injury

- Thermal injuries
- Electrical injury
  - High voltage
    - True high tension injury-current passes through patient
      - Voltage > 1000V
      - Extensive tissue damage
      - Complicated by rhabdomyolysis and renal failure
    - Flash-tangential exposure to high voltage current arc
      - Superficial burns to exposed body parts
      - Can set clothing alight

Mechanisms of injury

- Thermal injuries
- Electrical injury
- Chemical injury
  - Deep burns, significant coagulative necrosis
  - Alkaline burns tend to penetrate deeper than acidic
  - Immediate irrigation of effected tissues limits depth of the burn

Specific chemical burns and treatments

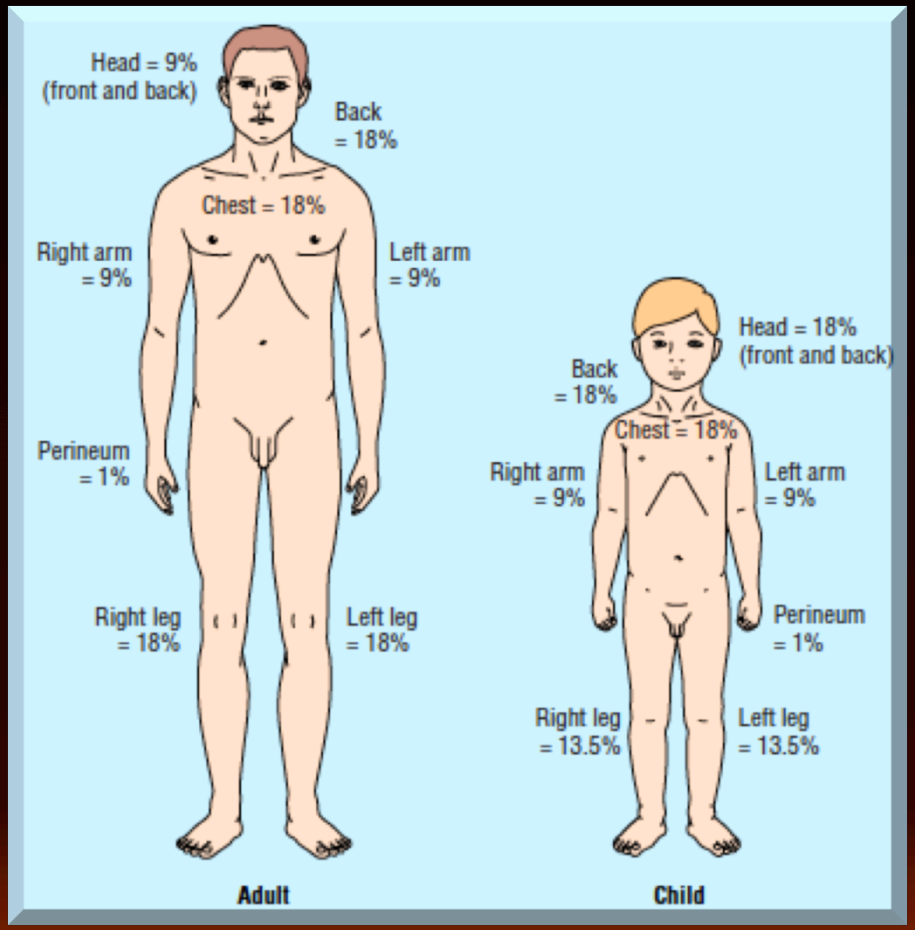
Chromic acid–Rinse with dilute sodium hyposulphite
Dichromate salts–Rinse with dilute sodium hyposulphite
Hydrofluoric acid–10% calcium gluconate applied topically as a gel or injected

Burn Area

- < 20% of TBSA are called local
- >20-30% of TBSA are called severe
- Local burns do not result in metabolic derangements
  - Aggressive systemic therapy is typically not required
- Methods of estimation
  - Palmar surface
  - Wallace Rule of Nines
  - Lund and Browder Chart

Wallace Rule of Nines

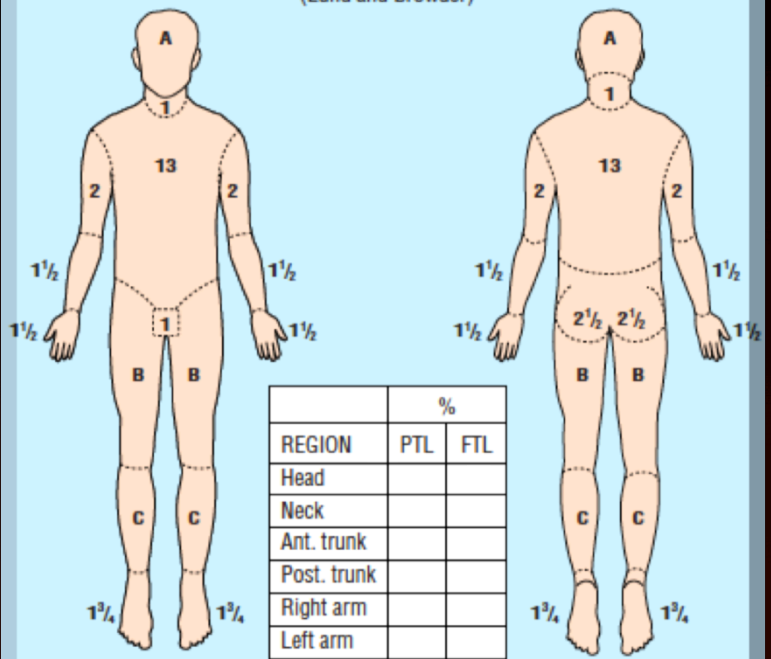
- Body is divided into areas of 9%
- Known to be inaccurate in children



Lund and Browder Chart

% Total Body Surface Area Burn

Be clear and accurate, and do not include erythema
(Lund and Browder)



	%	
REGION	PTL	FTL
Head		
Neck		
Ant. trunk		
Post. trunk		
Right arm		
Left arm		
Buttocks		
Genitalia		
Right leg		
Left leg		
Total burn		

AREA	Age 0	1	5	10	15	Adult
A – ½ OF HEAD	9½	8½	6½	5½	4½	3½
B – ½ OF ONE THIGH	2¾	3¼	4	4½	4½	4¾
C – ½ OF ONE LOWER LEG	2½	2½	2¾	3	3¼	3½

Treatments

- General therapies
  - Cooling
  - Fluid therapy
  - Pain management
  - Nutrition
- Specific wound management
  - Medical
  - Surgical

Cooling

- Benefits
  - Prevents ongoing tissue damage
  - Reduces edema formation
  - Increases epithelialization
  - Contributes to analgesic regimen
- Initiate within 30 minutes of injury
- 59° tap water for 20 minutes
- Avoid hypothermia

Fluid therapy

- Goal is to maintain perfusion to the zone of stasis
- Greatest amount of fluid loss in burn patients in 1st 24 hrs
  - More fluid is typically required for high tension electrical injury or inhalation injury
- First 8-12 hrs general shift from intravascular to interstitial
- Bolus therapy thought to be of little benefit

Fluid Creep

- Tendency to overuse crystalloid fluids with excessive initial resuscitation
- Prejudice against colloids
- Lack of attention to fluid volumes by clinicians
- Inaccurate estimation of total body surface areas
- Results in several of the complications we will talk about later

Resuscitation Formulae

- In human medicine, resuscitation is formula driven
- Most common is the Consensus or Parkland formula
- Crystalloid only

Parkland formula for burns resuscitation

Total fluid requirement in 24 hours =
4 ml×(total burn surface area (%))×(body weight (kg))
50% given in first 8 hours
50% given in next 16 hours
Children receive maintenance fluid in addition, at hourly rate of
4 ml/kg for first 10 kg of body weight *plus*
2 ml/kg for second 10 kg of body weight *plus*
1 ml/kg for >20 kg of body weight

End point
Urine output of 0.5-1.0 ml/kg/hour in adults
Urine output of 1.0-1.5 ml/kg/hour in children

Resuscitation Formulae

- Colloids
  - 0.5 mL x total burn surface area (%) x body weight (kg) and maintenance crystalloid is continued at 1.5 ml x (burn area) x (body weight)
- End point UOP:
  - 0.5-1 mL/kg/hr in adults
  - 1-1.5 ml/kg/hr in children

Resuscitation Formulae

- Hypertonic saline
  - Controversial
  - Reduces the amount of fluid need
  - Decreased risk of abdominal compartment syndrome
  - Increased risk of kidney injury and death
  - Requires very close monitoring of sodium levels

Pain management

- Fluctuates from day to day
- Opioid doses may exceed traditional dosing
- Benzodiazepines decrease background/procedural pain
- Ketamine or propofol have been shown to reduce procedural pain

Type of pain	Pain ptimulus	Characteristics
Procedural pain	Debridement	Burning and stinging with sharp pains
	Dressing changes	Persists for minutes to hours after dressing changes
	Hydrotherapy Physical therapy	
Background pain	Occurs at rest	Prolonged duration
	Normal daily activity	Relatively constant nature Continuous "burning" or "throbbing" Mild to moderate intensity
Breakthrough pain	Movement after long period of immobility	Occurs despite stable analgesic regimen Severe intensity Short duration Unpredictable

Pain management

- Degree of pain mediated by several factors

Factor	Relationship to pain intensity
Size of burn	Relationship unclear
Depth of burn	Superficial: initially acutely painful, recedes with healing
	Dermal and deeper: initial dull sensation, may develop increasing and complex pain
Location of burn	Worse in high mobility areas
Anxiety and depression	Direct relationship to pain perception
Inflammation	Inflammatory mediators modulate sensation
Long-term effects (scar and nerve)	Healing progression results in a wide range of complex neural abnormalities
Early grafting	Reduces incidence of chronic pain

Pain management

- Non-pharmacological therapies
  - Aromatherapy
  - Massage
  - Distraction therapy
  - TENS
  - Hypnosis

Nutrition

- Hypermetabolic response
  - Mediated by systemic response to burn
  - Related to the extent of the burn injury
  - Resting energy expenditure may be >100% basal expenditure
- Institution of enteral feeding should occur w/in 24-48 hours postburn injury

Management of the hypermetabolic response

- Reduce heat loss—environmental conditioning
- Excision and closure of burn wound
- Early enteral feeding
- Recognition and treatment of infection

Nutrition

- Antioxidants
  - No clinical data available
  - Vitamin C reduces fluid requirements in sheep
  - Currently recommended by ABA

Medical management

- All burn wounds should be cleansed
  - 1:40 dilution of chlorhexadine solution
  - 1:9 dilution of povidone iodine
  - 1-2 times daily, initially
  - Water cleansing with a handheld shower spray nozzle
- Variable recommendations based on burn depth

Epidermal burns

- Consider analgesia or other supportive measures
- Healing occurs rapidly, 3-5 days

Superficial partial thickness

- Expect healing in 2 weeks
- Deroof blisters by 2 days
- Aimed at preventing wound progression
- Topical antibiotics and occlusive dressings
  - Must be changed on alternate days

Deep partial thickness

- Slow to heal due to low density of skin adnexae
- More contraction is seen
- Extensive wounds or wounds over functionally important areas may be best managed by excision and grafting
- Will heal if optimized to encourage endogenous healing
  - Moist/warm
  - Free of infection
  - TransCyte

Full Thickness

- All regenerative elements destroyed
- Healing only occurs from the edge of the wound
- Associated with considerable contraction
- Should be excised and grafted

Type of excision	Technique	Utility	Advantages	Disadvantages
Tangential	Shave layers of devitalized tissue off until exposing a viable tissue bed (capillary bleeding noted)	Small burns	More cosmetic procedure	More blood loss
			Preserves body contours	Longer procedure time
Fascial	Complete excision of skin, subcutaneous fat, involved muscle fascia	Large, deep life-threatening burns	Faster procedure time	Severe cosmetic deformity
			Less skin grafting Less blood loss	Loss of cutaneous nerves

Timing of surgery

- Perform within 5 days to minimize blood and fluid loss, avoid inflammatory or infectious complications, and achieve best success
  - Large areas of burnt tissue may result in MOF or severe infections
  - Early excision results in reduction in the concentrations of Il-1, Il-6 and TNF-a
  - Eschar provides a good medium for bacterial growth
  - Exudation of fluid at the eschar worsens fluid, protein, immunoglobulin, and electrolyte losses

Escharotomy

- Indications
  - Circumferential deep dermal or full thickness wounds leading to high tissue pressures
  - Circumferential chest burns limiting chest excursion and impair ventilation
- Only separate burned tissue

Skin grafting

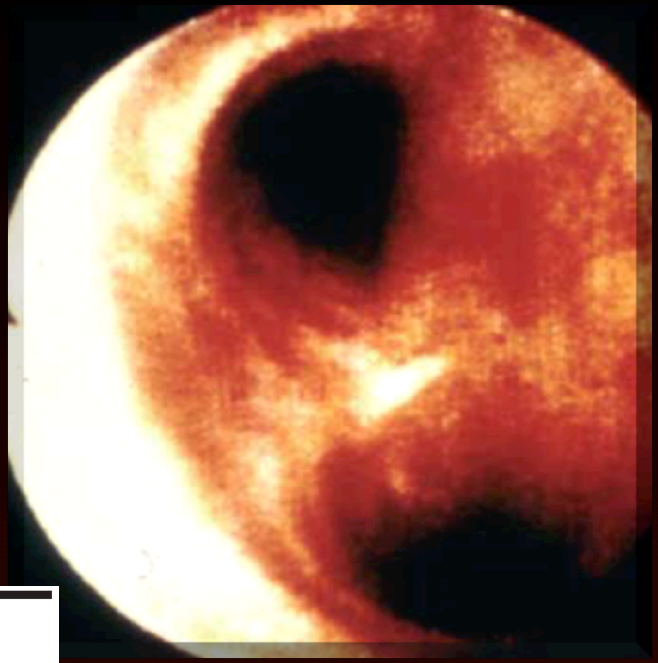
- Options:
  - Split skin autograft
  - Mesh graft
  - Rotation of donor sites
  - Temporary covering
    - Cadaveric allograft
    - Xenograft
    - Synthetic products
    - Cultured epithelial autograft

Complications

- Airway burns
- Heart failure
- Kidney injury
- Cerebral failure
- Ocular injury
- Infection
- Hypothermia
- Compartment syndrome

Airway burns

- Diagnosis based on PE and history
- ≥ 1 warning sign→high index of suspicion
- Clinical manifestation often delayed by 24hrs



Warning signs of airway burns

Suspect airway burn if:

- Burns occurred in an enclosed space
- Stridor, hoarseness, or cough
- Burns to face, lips, mouth, pharynx, or nasal mucosa
- Soot in sputum, nose, or mouth
- Dyspnoea, decreased level of consciousness, or confusion
- Hypoxaemia (low pulse oximetry saturation or arterial oxygen tension) or increased carbon monoxide levels (> 2%)

Onset of symptoms may be delayed

Airway burns

- Compromised pulmonary function is multifactorial
  - Upper airway obstruction
    - Airway control may be necessary
    - Swelling following resuscitation may lead to airway compromise

Mechanisms of pulmonary insult after lower airway burns

- Mucosal inflammation
- Mucosal burn
- Bronchorrhoea
- Bronchospasm
- Ciliary paralysis
- Reduced surfactant
- Obstruction by debris
- Systemic inflammatory response

Airway burns

- Compromised pulmonary function is multifactorial
  - Upper airway obstruction
  - Lower airway obstruction
    - Sloughing of tracheal membrane
    - Cast formation
    - Segmental atelectasis

Mechanisms of pulmonary insult after lower airway burns

- Mucosal inflammation
- Mucosal burn
- Bronchorrhoea
- Bronchospasm
- Ciliary paralysis
- Reduced surfactant
- Obstruction by debris
- Systemic inflammatory response

Airway burns

- Compromised pulmonary function is multifactorial
  - Upper airway obstruction
  - Lower airway obstruction
  - Decreased pulmonary compliance
    - Bronchospasm, atelectasis, and pulmonary edema

Mechanisms of pulmonary insult after lower airway burns

- Mucosal inflammation
- Mucosal burn
- Bronchorrhoea
- Bronchospasm
- Ciliary paralysis
- Reduced surfactant
- Obstruction by debris
- Systemic inflammatory response

Airway burns

- Compromised pulmonary function is multifactorial
  - Upper airway obstruction
  - Lower airway obstruction
  - Decreased pulmonary compliance
  - Reduced chest wall compliance
    - Torso burns
    - Pain
    - May require escharotomy to improve compliance

Airway burns

Ventilatory strategies to improve outcome have been proposed:

Possible ventilatory strategies for patients with airway burns

- Low volume ventilation
- Permissive hypercapnia
- High frequency percussive ventilation
- Nitric oxide
- Surfactant replacement
- Partial liquid ventilation (experimental)
- Extracorporeal membrane oxygenation (limited application)

Heart failure

- Myocardial dysfunction
  - Myocardial depressant factors→diastolic dysfunction
  - Myocardial edema
- Burn shock
- Electrical burn associated with arrhythmias
- Treatment
  - Correct underlying shock, as able
  - Inotropic drugs should not be used until adequate fluid resuscitation has occurred

Kidney Injury

- Early onset due to delayed or inadequate fluid resuscitation
- Delayed onset due to sepsis and associated multiorgan failure

Cerebral failure

- Hypoxic cerebral insult and closed head injury are associated with burn wounds
- Cyanide or carbon monoxide toxicities can result in forebrain signs

Ocular injury

- Multifactorial
  - Exposure keratopathy
  - Corneal ulceration
- Orbital compartment syndrome
  - Progressive periorbital tissue swelling alongside eyelid contracture
  - Results in increased intraocular pressure and optic neuropathy
  - Treatment by lateral canthotomy

Infection

- Contributory factors
  - Destruction of skin or mucosal surface barrier
  - Presence of necrotic tissue and exudate
  - Invasive monitoring devices
  - Impaired immune function
- Most common infections are pulmonary infections
  - 50-70% of patients with smoke inhalation develop pneumonia

Risk factors for pneumonia

- Inhalational injury:
  a) Destruction of respiratory epithelial barrier
  b) Loss of ciliary function and impaired secretion clearance
  c) Bronchospasm
  d) Mucus and cellular plugging
- Intubation
- Circumferential, full thickness chest wall burns
  Decreased chest wall compliance
- Immobility
- Uncontrolled wound sepsis
  Can lead to secondary pneumonia from haematogenous spread of organisms from wound

Infection

- Pathogenesis
  - Burn injury destroys most surface microbes
    - Not gram positive organisms in the hair follicles
  - Gram positive colonization at 48 hours
    - Minimized by the use of topical antibiotics
  - Gram negative bacteria conlonization at 3-21 days after injury
  - Invasive fungal infections seen later

Infection

- Prevention
  - Early aggressive surgery
  - Topical antimicrobials
    - Slows wound colonization
    - Of use in delayed definitive surgery
  - Systemic antibiotics is controversial
    - Generally agreed use of gram positive is not indicated
    - Broad spectrum antibiotics not indicated if wound covers <40% of total body surface area

Infection

Advantages and adverse effects of topical antimicrobials

Silver sulfadiazine

- Water soluble cream
- *Advantages*—Broad spectrum, low toxicity, painless
- *Adverse effects*—Transient leucopenia, methaemoglobinaemia (rare)

Cerium nitrate-silver sulfadiazine

- Water soluble cream
- *Advantages*—Broad spectrum, may reduce or reverse immunosuppression after injury
- *Adverse effects*—As for silver sulfadiazine alone

Silver nitrate

- Solution soaked dressing
- *Advantages*—Broad spectrum, painless
- *Adverse effects*—Skin and dressing discoloration, electrolyte disturbance, methaemoglobinaemia (rare)

Mafenide

- Water soluble cream
- *Advantages*—Broad spectrum, penetrates burn eschar
- *Adverse effects*—Potent carbonic anhydrase inhibitor—osmotic diuresis and electrolyte imbalance, painful application

Infection

- Diagnosis
  - Wound biopsy
  - Clinical evaluation
    - Local signs
    - Systemic signs
      - Alteration of mental status
      - Worsening pulmonary function
      - Impaired renal function
      - Intolerance of enteral feedings
      - Persistent hyperglycemia
- Treatment
  - Antibiotics +/- debridement

Signs of wound infection

- Change in wound appearance:
  a) Discoloration of surrounding skin
  b) Offensive exudate
- Delayed healing
- Graft failure
- Conversion of partial thickness wound to full thickness

Hypothermia

- Potential adverse effects
  - Hypocoagulability
  - Altered drug metabolism
  - Increased oxygen consumption
  - Increased risk of infection
- Various methods can be used to minimize its incidence

Compartment syndrome

- Extremity compartment syndrome
- Abdominal compartment syndrome
  - Decreased abdominal wall compliance from circumferential wounds
  - As IAP increases, hypertension ensues
  - Can result in pressure-induce organ dysfunction
  - Non-invasive therapies are suggested in IAP increases above 20 cm H2O in dogs and cats

Improve compliance
Sedation and analgesia to decrease thoracoabdominal muscle tone
Perform escharotomy on circumferential torso burns
Alter gastrointestinal motility and volume
Prokinetics (eg, erythromycin or metoclopramide)
Nasogastric drainage
Enemas
Evacuate intraperitoneal fluid
Percutaneous catheter abdominal decompression

Prognostication

- Increased risk of death with
  - Increasing age
  - Increasing burn size
  - Presence of inhalational injury