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 salvageable
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/thromboxananes
    - 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
• 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
Resuscitation 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

<table>
<thead>
<tr>
<th>Classification of burn wound</th>
<th>Dermal layers involved</th>
<th>Wound characteristics</th>
<th>Healing</th>
</tr>
</thead>
</table>
| Superficial                  | Epidermis only         | Erythematous desquamation  
|                              |                        | Dry, flaky appearance   | Heals in 3–5 days via re-epithelialization  
|                              | Epidermis              | Erythematous, moist blanches | Minimal scar formation |
|                              | Upper 1/3 of dermis    | Painful blisters may be present | Heals in 1–2 weeks via re-epithelialization |
|                              | (papillary layer)      | Edema may be present  
|                              |                        | Eschar formation        | Minimal scar formation |
| Deep partial-thickness       | Epidermis              | Red-waxy white        | Heals in 2–3 weeks      |
|                              | All dermis             | Reduced pain sensation | Recommend surgical intervention to prevent significant scar formation |
|                              |                        | Blisters absent       |                     |
|                              |                        | Eschar formation      |                     |
| Full-thickness               | Epidermis dermis       | Bloodless pearl-white Eschar formation | Requires surgical intervention |
|                              | Subcutaneous tissue    | Hair easily plucked   |                     |
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 = \[0.24 \times (\text{voltage})^2 \times \text{resistance}\]
    - 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
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)

<table>
<thead>
<tr>
<th>REGION</th>
<th>PTL</th>
<th>FTL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neck</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ant. trunk</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post. trunk</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right arm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left arm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Buttocks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genitalia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right leg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left leg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total burn</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>AREA</th>
<th>Age 0</th>
<th>1</th>
<th>5</th>
<th>10</th>
<th>15</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>A = % OF HEAD</td>
<td>0%</td>
<td>8%</td>
<td>6%</td>
<td>5%</td>
<td>4%</td>
<td>3%</td>
</tr>
<tr>
<td>B = % OF ONE THIGH</td>
<td>2%</td>
<td>3%</td>
<td>4%</td>
<td>4%</td>
<td>4%</td>
<td>4%</td>
</tr>
<tr>
<td>C = % OF ONE LOWER LEG</td>
<td>2%</td>
<td>2%</td>
<td>2%</td>
<td>3%</td>
<td>3%</td>
<td>2%</td>
</tr>
</tbody>
</table>
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 =

\[
4 \text{ ml} \times (\text{total burn surface area } (\%)) \times (\text{body weight } (\text{kg}))
\]

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 \textit{plus}
- 2 ml/kg for second 10 kg of body weight \textit{plus}
- 1 ml/kg for \( > 20 \) kg of body weight \textit{plus}

**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

<table>
<thead>
<tr>
<th>Type of pain</th>
<th>Pain stimulus</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procedural pain</td>
<td>Debridement</td>
<td>Burning and stinging with sharp pains</td>
</tr>
<tr>
<td></td>
<td>Dressing changes</td>
<td>Persists for minutes to hours after dressing changes</td>
</tr>
<tr>
<td></td>
<td>Hydrotherapy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Physical therapy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Occurs at rest</td>
<td></td>
</tr>
<tr>
<td>Background pain</td>
<td>Normal daily activity</td>
<td>Relatively constant nature</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Continuous “burning” or “throbber”</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mild to moderate intensity</td>
</tr>
<tr>
<td>Breakthrough pain</td>
<td>Movement after long period of</td>
<td>Occurs despite stable analgesic regimen</td>
</tr>
<tr>
<td></td>
<td>immobility</td>
<td>Severe intensity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Short duration</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Unpredictable</td>
</tr>
</tbody>
</table>
Pain management

- Degree of pain mediated by several factors

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

<table>
<thead>
<tr>
<th>Management of the hypermetabolic response</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Reduce heat loss—environmental conditioning</td>
</tr>
<tr>
<td>• Excision and closure of burn wound</td>
</tr>
<tr>
<td>• Early enteral feeding</td>
</tr>
<tr>
<td>• Recognition and treatment of infection</td>
</tr>
</tbody>
</table>
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

<table>
<thead>
<tr>
<th>Type of excision</th>
<th>Technique</th>
<th>Utility</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tangential</td>
<td>Shave layers of devitalized tissue off until exposing a viable tissue bed (capillary bleeding noted)</td>
<td>Small burns</td>
<td>More cosmetic procedure</td>
<td>More blood loss</td>
</tr>
<tr>
<td>Fascial</td>
<td>Complete excision of skin, subcutaneous fat, involved muscle fascia</td>
<td>Large, deep life-threatening burns</td>
<td>Preserves body contours</td>
<td>Longer procedure time</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Faster procedure time</td>
<td>Severe cosmetic deformity</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Less skin grafting</td>
<td>Loss of cutaneous nerves</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Less blood loss</td>
<td></td>
</tr>
</tbody>
</table>
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
- $\geq 1$ warning sign $\rightarrow$ 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

<table>
<thead>
<tr>
<th>Mechanisms of pulmonary insult after lower airway burns</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mucosal inflammation</td>
</tr>
<tr>
<td>Mucosal burn</td>
</tr>
<tr>
<td>Bronchorrhoea</td>
</tr>
<tr>
<td>Bronchospasm</td>
</tr>
</tbody>
</table>
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:

- 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:
  - Destruction of respiratory epithelial barrier
  - Loss of ciliary function and impaired secretion clearance
  - Bronchospasm
  - 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 colonization 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
## 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-induced organ dysfunction
  - Non-invasive therapies are suggested in IAP increases above 20 cm H2O in dogs and cats

<table>
<thead>
<tr>
<th>Improve compliance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedation and analgesia to decrease thoracoabdominal muscle tone</td>
</tr>
<tr>
<td>Perform escharotomy on circumferential torso burns</td>
</tr>
</tbody>
</table>

**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