Thermal Burns

• Epidemiology
  – Rank third among injury related deaths in kids aged 1-9
  – Pediatric and elderly patients have the highest morbidity and mortality
  – Approximately 80,000 hospitalizations each year
    • 1/3 – ½ are younger than 18
  – Birth to age 4 account for ~50% of pediatric burns
• Most common types in childhood
  – Flames
  – Scalds
  – Contact
  – Cold
  – Radiation
• In toddlers, scalds account for 80% of thermal injuries
• Toddlers also have the highest rate of contact burns
• Young school age kids play with fire
• Older kids take risks with fireworks and gasoline
• More common in boys than girls
• **Pathophysiology**
  – Skin serves to:
    • Protect from infectious agents
    • Regulate body temperature
    • Barrier against fluid loss
  – Skin consists of two layers, the epidermis and dermis
    • Epidermis has four layers
      – Stratum corneum – most important layer in protection against water loss and infection
      – Stratum lucidum
      – Stratum granulosum
      – Stratum germinativum
    • Dermis consists of hair follicles, nerve endings and connective tissue
• **Pathophysiology**
  - With deep burns, there may be a clear cut area of irreversible necrosis
  - Surrounding that, an area of ischemia
    • Tissue here may survive or die depending on preservation of blood flow
  - Surrounding the ischemia zone is a zone of hyperemia
    • Increased blood flow promoted by numerous mediators liberated from injured tissues
• Severe burns of >25% TBSA
  – Noninjured tissues will also swell secondary to presence of various mediators

<table>
<thead>
<tr>
<th>Mediator</th>
<th>Source</th>
<th>Effect</th>
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<tbody>
<tr>
<td>Histamine</td>
<td>Mast cells from burned skin</td>
<td>Increases capillary permeability, arteriolar dilatation, and venular contraction</td>
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<tr>
<td>Prostaglandins</td>
<td>Arachidonic acid released from burned tissue and inflammatory cells</td>
<td>PGE₂, PGI₂; potent vasodilators; increase microvascular permeability</td>
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<tr>
<td>Thromboxanes</td>
<td>Platelets in the burn wound</td>
<td>Thromboxanes A₂ and B₂; vasconstrictors; contribute to tissue ischemia</td>
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<tr>
<td>Kinins</td>
<td>Inflammatory cells</td>
<td>Increase venular permeability</td>
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<tr>
<td>Serotonin</td>
<td>Inflammatory cells</td>
<td>Vasoconstrictor; reduces blood flow to burn wounds</td>
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<tr>
<td>Catecholamines</td>
<td>Adrenal medulla</td>
<td>Vasoconstrictor; contributes to wound ischemia, increased systemic vascular resistance</td>
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<tr>
<td>Oxygen radicals</td>
<td>Burned tissue</td>
<td>Vasoconstrictor; may be responsible for intestinal ischemia (angiotensin) and increased systemic vascular resistance (vasopresin)</td>
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<tr>
<td>Platelet aggregation factor</td>
<td>Burn wound platelets</td>
<td>Vasoconstrictor; increases capillary permeability and burn edema</td>
</tr>
<tr>
<td>Angiotensin II and vasopresin</td>
<td>Renal juxtaglomerular cells</td>
<td>Vasoconstrictor; increases capillary permeability</td>
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</table>
– These mediators impair cardiac contractility and increase vascular resistance
  • Sets a scene for
    – Hypovolemia
    – Hypoperfusion
    – Tissue ischemia
    – Renal failure
    – SIRS
• Classification
  – Thickness of a burn is directly related to the source of burn and time in contact
  – First degree
    • Erythematous and painful
    • Involve epidermis without blistering
    • Heal within 4-5 days without scarring
    • E.g. sunburn
• Classification
  – Second degree/partial thickness
    • Superficial partial thickness burns
      – involve partial destruction of dermis
      – Red, weeping, blistered and painful
      – Heal in 7-10 days with minimal scarring
• Deep partial thickness burns
  – Involve greater than 50% of dermis, destroying nerve fibers
  – White, pale appearance, and are less painful
  – >2-3 weeks to heal, and usu req grafting for long term
  – Patients with significant burns at risk for fluid loss
• **Classification**
  – **Full thickness/third degree burns**
    • White, waxy or leathery and do not bleed, painless
    • At high risk for infection and fluid loss
    • Several weeks to heal and scar significantly
  – **Fourth degree burns**
    • Not commonly used terminology
    • Involve destruction of underlying structures like fascia, tendons, muscle and bone
    • Mostly seen with severe electrical injury
• Extent of Burn
  – “Rule of Nines”
  – Superficial burns should not be included in calculation as they do not affect fluid loss
  – In adults:
    • Head and each arm are 9% of TBSA each
    • Anterior and posterior trunk and each leg are 18% TBSA each
    • Neck and groin are 1% TBSA each
– In kids
  • Area of child’s palm is ~1% TBSA
• Initial assessment
  – ABC’s
  – Determination of burn depth
  – TBSA involvement
    • Circumferential burns noted, as they can lead to compartment syndrome and require escharotomy
      – Circumferential burns of the chest may interfere with ventilation
    • Remove all clothing
    • Apply saline soaked gauze/sheet to wounds
      – Decreases environmental exposure and pain
• Labs
  – CBC and chemistries
    • Baseline values, as pt may soon experience major fluid shifts and changes in metabolic status
  – U/A
    • Assess for myoglobin, which can lead to renal function impairment
• Airway/Breathing
  – House fires, indoor fires, and chemical fires may involve respiratory tract burns resulting in inflammation and edema
  – Anticipate airway compromise with
    • Stridor, hoarseness, carbonaceous sputum, perioral or perinasal burns
    • Intubate
      – Airway edema may not be apparent until 48 hours after a burn, and, if you wait….difficult intubation
      – Anticipate a narrowed airway, and have smaller ETT available
        » Supraglottic injury usually a result of direct thermal injury
        » Lower airway edema a result of chemicals such as smoke, and leads to chemical pneumonitis
• Ventilation
  – Is high PEEP low volume ventilation in burn patients beneficial? Burns, 2004
    • Retrospective study of 61 patients
  – Inhalation injury increases mortality up to 40% in combination with a severe burn
  – If thermal injury precedes smoke inhalation, lung damage is less severe than vice versa
    • Mechanism?
  – Inhalation injury treated with mechanical ventilation
• Ventilation
  - In inhalation injury, many of the conditions of ARDS and VILI are induced by initial insult
    • Cellular integrity disrupted
    • Cellular function altered
    • Blood flow regulation altered
    • Capillary leak
  - High PEEP, low volume ventilation helps with decreasing incidence of pulm edema, but no difference in mortality seen
• All victims of house or indoor fires should be evaluated for CO poisoning
• Circulation
  – A burn that is 15-20% TBSA will result in hypovolemic shock
    • “burn shock” results from system wide extravasation of fluids into unburned tissues
    • This is coupled with increased evaporative water losses
  – Replace with isotonic fluids, not albumin
    • Capillary leak leads to extravasation of albumin, increasing oncotic pressure in interstitium, and increasing extravasation of fluid
• Circulation
  – Parkland Formula:
    • 4 ml x TBSA x weight (kgs)
    • Half of total fluids given in first 8 hours, next ½ over 16 hours
      – Done slowly secondary to severe capillary leak present; increased fluids will increase total body and wound edema because of increased hydrostatic pressure in the face of lower oncotic pressure
        » Titrate for UOP ~ 1cc/kg/hr
        » Pulmonary edema can develop rapidly
      – Added to maint. requirements
    • Warm fluid
      – As pt is at risk for hypothermia
• Triage
  – Admit to hospital if:
    • Partial thickness burns 10-20% TBSA
    • Full thickness burns 5% TBSA
  – Burn Center
    • Partial thickness burn > 20% TBSA in any age
      – Or >10% in kids <10yrs of age
    • Full thickness burns >5%
    • Burns to face, hands, feet, genitalia, major joints
    • Inhalation burns
    • Electrical burns
  – All others OK for outpt tx.
• **Treatment**
  
  – **Pain control**
    • Cover burns
    • NSAIDs, narcotics
  
  – **Clean burns**
    • Unroof blisters, never aspirate
    – Burn fluid
      » contains cytokines that suppresses neutrophil and lymphocyte response
      » Interferes with fibrinolysis
      » Increase inflammatory response which increases infectious risk
      » Thromboxanes promote dermal ischemia, leading to progression of burn depth
      » Great culture medium for bacteria
• **Treatment**
  – All burns at increased risk for infection
    • Tetanus vacc should be given, if needed
  – NO role for initial antibiotic therapy
  – Topical creams are preferred for burns
    • Decrease water loss
    • Contribute to pain control
    • Inhibit bacterial and fungal growth
    • Keep area safe from dessication
• Silver Sulfadiazine 1%
  – Painless and bactericidal, good for Gm + orgs
  – May stain skin, ineffective against Pseudomonas and leads to hyponatremia

• Mafenide Acetate
  – Carbonic anhydrase inhibitor
  – Bacteriostatic with good pseudomonal coverage
  – Penetrates eschar
  – Painful and may lead to metabolic acidosis
• Treatment
  – Elevate extremity
    • Failure to do so leads to increased edema, which will compromise blood flow to ischemic areas
  – Dressings
    • Amniotic membrane
    • Keratinocyte culture
    • Artificial skin
    • Human allograft
    • Pigskin
    • Dressing adheres to the wound until epithelialization occurs
• Treatment
  – Nutritional support
    • Catabolic response is severe, and BMR may be >2x baseline
    • Metabolic requirement increases with extent of burn
    • High catecholamines, cortisol and glucagon antagonize effects of insulin and GH
      – Body less able to use fats, muscle becomes energy source
    • High fat intake deleterious
      – Fatty acids, in their oxidized form, are inflammatory to cells
      – Systemic oxidants elevated in burn patients and endogenous antioxidants are low
• Morbidity
  – Burn patients die for three main reasons
    • Burn shock during the first few hours
    • Respiratory failure in the following days
    • Septic complications and SIRS in following weeks
  – The first two reasons are now rare thanks to advances in fluid management and ventilator support
  – This has led to increased prevalence of infection as a cause of late mortality
    • Infection is now the biggest killer in the burn unit
• **Infection**
  - Rates are similar to other groups of immunocompromised patients
  - Increased infection susceptibility secondary to
    • Local factors
      – Open wounds
      – Incompetent gut barrier
      – Exposure of cartilage, bone and joints
    • Systemic factors
      – Global decrease in cellular immune function
      – Neutropenia is common, and neutrophil function is depressed
      – Increased gut permeability
      – Occult bacteremia occurs with wound manipulation
• Infection
  – Difficult to recognize
    • Typical burn exhibits inflammation along with erythema, tenderness and edema
    • Burns, secondary to widespread mediator release, are also associated with fever, in the absence of infection
    • Infection of wounds may lead to sepsis and to deeper wound damage
Burn wound infection
Intercompartmental infection
• Infection
  – Broad spectrum prophylactic antibiotics contraindicated
    • Promote fungal growth
  – Antistreptococcal antibiotics
    • Contraindicated since wound excision and closure practice combined with topical creams has dramatically reduced wound infection rates
• Survival benefit in Critically Ill burn patients receiving selective Contamination of the Digestive Tract, Annals of Surgery, March 2005
  – 107 pts
    • > 14yrs old, with burns of >20% TBSA
  – SDD included
    • IV cefotaxime for four days
    • Topical oropharyngeal polymixin, tobra and ampho
    • 10ml solution of polymyxin, tobra and ampho QID
  – Reduction in mortality of 57% in burn ICU and 50% in hospital mortality
  – Reduction in primary endogenous infections, PNAs and UTIs due to community bacteria
  – No difference in incidence of secondary endogenous infections due to hospital bacteria
• Healing
  – Occurs by epithelialization, which begins in hair follicles that remain in dermis
  – Emerging epithelial buds grow together to close the wound
  – Full thickness wounds destroy follicles, so grafts must be used
• Scarring may be extensive
• **Nonaccidental Burns**
  - 10-20% of burns in kids are inflicted
  - 16-20% of children admitted to hospitals with burns are victims of abuse

• **Have recognizable patterns or linear lines of demarcation**

• **Severe burns to hands and feet in stocking glove pattern is classic**
Electrical Burns

• Epidemiology
  – Result in over 1500 deaths per year
  – Up to 1/3 of electrical burns are household burns, seen mostly among children
Pathophysiology

- Electrical burns result from thermal energy produced as current passes through the body
- Thermal energy produced is proportional to current
- Extent of injury depends on:
  - Resistance of skin, mucosa and internal structures
  - Type of current
  - Frequency of current
  - Duration of contact
  - Intensity of current
  - Pathway taken by current
• Resistance
  – Is inversely proportional to tissue injury
  – Nerves, muscles and blood vessels have low resistance
    • Current passes through these and causes damage
  – Water will decrease resistance
    • Results in moist areas of body, like the axilla, sustaining greater injury
• Type of current
  – AC more dangerous than DC
    • AC produces muscle tetany caused by continual contraction and relaxation with each cycle
    • Typically found in household electricity
    • 60Hz current changes 120 times per sec
      – Prevents muscle relaxation and keeps it in a continual refractory state
      – If it happens to chest wall muscles, suffocation occurs
      – If a patient is holding on to current, can’t let go
    • DC current found in medical settings and is found in lightning strikes
      – At risk for VFib or asystole
• Voltage
  – Low current injuries
    • Young kids putting electrical cord in their mouth
  – Medium current and High current
    • Seen in adolescents with risk taking behavior
      – Lightning strikes and climbing electrical poles
• Path of Current
  – Current will flow from point of contact to the ground or part of the body that completes the circuit
  – Hand to hand flow
    • 60% mortality
      – spinal cord transection at C4-C8
      – suffocation by way of chest wall tetany
      – myocardial damage
• Path of Current
  – Hand to Foot
    • 20% mortality
      – Cardiac arrhythmias
  – Foot to Foot
    • Less than 5% mortality
• Assessment
  – ABC’s
    • Cardiac monitoring secondary to arrythmias
      – VFib seen in low voltage and AC injuries
      – High voltage injuries produce asystole
  – Minor superficial injury may mask significant underlying tissue damage
    • Consider head, spine and abdominal CT’s
• Triage
  – Low voltage injuries
    • Ok for home after 4 hours of ED cardiac monitoring
  – Medium and high voltage injuries
    • Admit to hospital for at least 48-72hrs of cardiac monitoring
    • Watch for myoglobinuria
      – Alkalinize urine, and increase UOP
Chemical Burns

- Between 25,000 and 100,000 burns in US each year
- Morbidity and mortality of less than 1%
- Children and adults with similar rates of exposure
• Acid burns
  – Result in coagulation necrosis
    • coagulation of proteins with some retention of cell architecture
  – Drain cleaners (sulfuric or hydrochloric acid)
  – Toilet cleaners (hydrochloric or phosphoric)
  – Car batteries (sulfuric acid)
• Alkali Burns
  – Produce liquefactive necrosis
    • No recognizable cellular architecture left
  – Lye (NaOH)
  – Cement (K, Ca and NaOH)
  – Oven and drain cleaners
• Treatment
  – Remove all clothes
  – Irrigation for 30min
    • DO NOT neutralize burn
      – Exothermic reaction may produce bad thermal injury
  – If ingested, charcoal is contraindicated as it does nothing to neutralize substance and obscures endoscopy
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