CHAPTER 8
THERMAL INJURIES

Thermal destruction of the skin results in severe local and systemic alterations. This destruction can occur from thermal energy, chemical reactions, electricity, or the response to cold. The management of the patient with a major thermal injury requires understanding of the pathophysiology, diagnosis, and treatment not only of the local skin injury but also of the derangements that occur in hemodynamic, metabolic, nutritional, immunologic, and psychologic homeostatic mechanisms.

I. BURNS

A. Pathophysiology: Amount of tissue destruction is based on temperature (>40˚C) and time of exposure (Fig. 8-1)

B. Diagnosis and prognosis

1. Burn size: % of total body surface area (TBSA) burned
   a. Rough estimate is based on rule of 9s (Fig. 8-2)
   b. Different charts are required for adults and children because of head-chest size discrepancy and limb differentials for ages birth to seven years (Fig. 8-3 and 8-4)
Fig. 8-2

Fig. 8-3
2. Age: burns at the extremes of age carry a greater morbidity and mortality

3. Depth: difficult to assess initially
   a. History of etiologic agent and time of exposure helpful
   b. Classification (Fig. 8-5)
      i. First degree: erythema but no skin breaks
      ii. Second degree: blisters, red and painful
         (a) Superficial partial-thickness, involves epidermis and upper dermis
         (b) Deep partial-thickness, involves deeper dermis
      iii. Third degree: full-thickness-insensate, charred or leathery
      iv. Fourth degree: muscle, bone

4. Location: face and neck, hands, feet, and perineum may cause special problems and warrant careful attention; often necessitate hospitalization/burn center

5. Inhalation injury: beware of closed quarters burn, burned nasal hair, carbon particles in pharynx, hoarseness, conjunctivitis

6. Associated injuries, e.g. fractures
7. Co-morbid factors, e.g. pre-existing cardiovascular, respiratory, renal and metabolic diseases; seizure disorders, alcoholism, drug abuse

8. Prognosis: best determined by burn size (TBSA) and age of patient, inhalation injury

9. Circumferential burns: can restrict blood flow to extremity, respiratory excursion of chest and may require escharotomy

C. Categorization of burns is used to make treatment decisions and to decide if treatment in a burn center is necessary (Table 8-1, Table 8-2)

D. Treatment plan
1. History and physical exam
2. Relieve respiratory distress — escharotomy and/or intubation
3. Prevent and/or treat burn shock — IV — large bore needle
4. Monitor resuscitation — Foley catheter and hourly urine output
5. Treat ileus and nausea — N.G. tube if > 20% burn
6. Tetanus prophylaxis
7. Baseline laboratory studies i.e. Hct., UA, glucose, BUN, chest x-ray, electrolytes, EKG, cross-match, arterial blood gases, and carboxyhemoglobin
8. Cleanse, debride, and treat the burn wound

E. Respiratory distress
1. Three major causes of respiratory distress in the burned patient:
   a. Unyielding burn eschar encircling chest
      i. Distress may be apparent immediately
      ii. Requires escharotomy (cutting into the eschar to relieve constriction)
   b. Carbon monoxide poisoning
      i. May be present immediately or later
      ii. Diagnosed by carboxyhemoglobin levels measured in arterial blood gas
      iii. Initial Rx is displacement of CO by 100% O2 by facemask
      iv. Hyperbaric oxygen treatment may be of value

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<th>Categorization of burns (American Burn Association):</th>
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<td>Size-Partial thickness</td>
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<td>Size-Full thickness</td>
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<tr>
<td>Primary areas</td>
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<td>Inhalation injury</td>
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<td>Associated injury</td>
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<td>Miscellaneous</td>
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<th>Burns That Dictate Patient Admission to a Hospital or Burn Center</th>
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<tr>
<td>1. 2˚ and 3˚ burns greater than 10% of BSA in patients under 10 or over 50 years of age</td>
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<td>2. 2˚ and 3˚ burns greater than 20% BSA in any age group</td>
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<td>3. 2˚ and 3˚ burns posing a serious threat of functional or cosmetic impairment, e.g. the face, hands, feet, genitalia, perineum, and about major joints</td>
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<td>4. 3˚ burns greater than 5% BSA in any age</td>
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<td>5. Electrical burns including lightning</td>
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<td>6. Chemical burns posing a serious threat of functional or cosmetic impairment</td>
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<td>7. Inhalation injury</td>
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<td>8. Burns associated with major trauma</td>
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Table 8-1

Table 8-2
c. Smoke inhalation leading to pulmonary injury
   i. Insidious in onset (18-36) hours
   ii. Due to incomplete products of combustion, not heat
   iii. Causes chemical injury to alveolar basement membrane and pulmonary edema
   iv. Initial Rx is humidified O₂ but intubation and respiratory support may be required
   v. Secondary bacterial infection of the initial chemical injury leads to progressive pulmonary insufficiency
   vi. Severe inhalation injury alone or in combination with thermal injury carries a grave prognosis
   vii. Three stages of presentation have been described:
       (a) Acute pulmonary insufficiency (immediately post burn to 48 hours)
       (b) Pulmonary edema (48-72 hours)
       (c) Bronchopneumonia (25 days)

F. Burn shock
   1. Massive amounts of fluid, electrolytes, and protein are lost from circulation almost immediately after burning (Table 8-3)

Burn or Associated Condition Dictating Extra Fluid Administration

- Underestimation of the %TBSA burn
- Burn greater than 80% TBSA
- Associated traumatic injury
- Electrical burn
- Associated inhalation injury
- Delayed start of resuscitation
- 4º burn
- Administration of osmotic diuretics
- Pediatric burns

Table 8-3

2. Resuscitation requires replacement of sodium ions and water to restore plasma volume and cardiac output
   a. Many formulas have been reported to achieve resuscitation
      i. This can be given by prescribing 4cc Ringer's lactate/Kg/%TBSA burn over the first 24 hours (Baxter or Parkland Hospital formula)
      ii. 1/2 of the first 24 hour fluid requirement should be given in the first eight hours postburn and the remaining 1/2 over the next 16 hours
   b. A plasma volume gap may remain
      Restored between 24-30 hours postburn by administering .35-.50cc plasma/Kg/% TBSA burn
   c. After 30 hours D5W can be given at a rate to maintain a normal serum sodium

G. Monitoring resuscitation
   1. Urine output 30-55cc/hr in adults and 1.2cc/Kg/hr in children < age 12
   2. A clear sensorium, pulse <120/min, HCO₃ > 18 meq/L, cardiac output >3.1 L/M²
   3. CVP in acute major burns is unreliable

H. Treatment of the burn wound (Table 8-4)
   1. Wound closure by the patient's own skin is the ultimate goal of treatment
      a. By spontaneous healing
      b. Autograft
      c. Allograft
      d. Xenograft
      e. Artificial skin
      f. Cultured epithelial cells
   2. Specific treatment of the burn wound differs from one burn center to another
      a. The most commonly employed topical antibacterials are silver sulfadiazine (Silvadene®) and mafenide acetate (Sulfamylon®)
      b. Status of burn wound bacterial colonization and effectiveness of topical antibacterial treatment can be monitored by biopsies of wound for quantitative and qualitative bacteriology
3. Necrotic tissues may be removed by any of several techniques:
   a. Formal excision
   b. Tangential (layered) debridement
   c. Enzymatic debridement
   d. Hydrotherapy — a useful adjunct
4. Autografts should be applied to priority areas first, such as the hands, face and important joints
5. Once healed, pressure is usually necessary with elastic supports to minimize hypertrophic scarring
6. Physical therapy — important adjunct in burn care
I. Complications: can occur in every physiologic system secondary to burn injury (Table 8-5)
   1. Renal failure
      a. From hypovolemia
      b. Beware of nephrotoxic antibiotics in the burn patient

Sample Orders
For a 70 Kg 40 year old patient with a 40% flame burn:
1. Admit to ICU portion of burn center
2. Strict bedrest with head elevated 45°
3. Maintain elevation of burned extremities
4. Vital signs; pulse, BP respiration q 15 min, temperature q 2 h
5. Check circulation of extremities (capillary refill or Doppler) q 30 min
6. 100% O₂ face mask
7. Infuse Ringer's lactate at 700cc for first hour, then reassess
8. Measure urinary output by Foley catheter to closed drainage
9. Notify physician of first hour's urine output (must be 30-50cc: 1.2-1.5cc in pediatric patient)
10. N.P.O.
11. N.G. tube to intermittent low suction
12. Measure pH of gastric content q 2 h — stress ulcer prophylaxis (e.g. Zantac)
13. Morphine sulfate 4 mg intravenously q 2-3 hr prn pain - no intramuscular narcotics (unreliable absorption)
14. Tetanus toxoid 0.5cc IM (if patient previously immunized)
15. Send blood for Hct., glucose, BUN, cross match 2 units, electrolytes
16. Urine for U.A. and culture
17. Chest x-ray
18. EKG
19. Arterial blood gases q 6 h and prn
20. Cleanse wounds with Betadine solution, debride all blisters, map injury on Lund-Browder chart, and photograph wounds
21. Apply silver sulfadiazine to all wounds with sterile gloved hand (use reverse isolation technique when burn wounds are exposed)
22. Dress wounds with burn gauze and surgifix
23. Splint extremities as per physical therapist
24. Change all dressings, cleanse wounds, and reapply topical antibacterial q 8 h or q 12 h
25. Bronchoscopy — If inhalation injury suspected

Table 8-4

Risk Factors in Burn Wound Infection

I. PATIENT FACTORS
   A. Extent of burn > 30% of body surface
   B. Depth of burn: full-thickness vs. partial-thickness
   C. Age of patient (very young or very old at higher risk)
   D. Pre-existing disease
   E. Wound dryness
   F. Wound temperature
   G. Secondary impairment of blood flow to wound
   H. Acidosis
II. MICROBIAL FACTORS
   A. Density >10⁵ organisms per gram of tissue
   B. Motility
   C. Metabolic products
      1. Endotoxin
      2. Exotoxins
      3. Permeability factors
      4. Other factors
   D. Antimicrobial resistance

Table 8-5
2. Gastrointestinal bleeding  
a. More likely in burns over 40%  
b. Usually remains subclinical  
c. Antacids and H₂ blockers  
d. Increased risk with burn wound sepsis  
3. Burn wound sepsis  
a. Monitored by tissue biopsy — qualitative and quantitative  
b. Must keep bacterial count < 10⁷ bacteria/gm of tissue  
c. Clinically suspect sepsis with  
   i. Sudden onset of hyper or hypothermia  
   ii. Unexpected congestive heart failure or pulmonary edema  
   iii. Development of the acute respiratory distress syndrome  
   iv. Ileus occurring after 48 hours postburn  
v. Mental status change  
vi. Azotemia  
vii. Thrombocytopenia  
viii. Hypofibrinogenemia  
ix. Hyper or hypoglycemia is especially suspect if burn > 40% TBSA  
x. Blood cultures may be positive but in many cases are not  
4. Progressive pulmonary insufficiency  
a. Can occur after:  
   i. Smoke inhalation  
   ii. Pneumonia  
   iii. Cardiac decompensation  
   iv. Sepsis from any cause  
b. Produces:  
   i. Hypoxemia  
   ii. Hypocarbia  
   iii. Pulmonary shunting  
   iv. Acidosis  
5. Wound contracture and hypertrophic scarring  
a. Largely preventable  
b. Since a burn wound will contract until it meets an opposing force, splinting is necessary from the outset  
   i. Splints are used to prevent joint contractures, e.g. elbow and knee are kept in extension, and MCP joints of fingers in flexion  
c. Timely wound closure with adequate amounts of skin should largely eliminate these problems  
d. Continued postoperative splinting and elastic pressure supports are of value in the remodeling of collagen with prevention of hypertrophic scars  

II. CHEMICAL BURNS  
A. Pathophysiology  
1. Tissue damage secondary to a chemical depends on:  
   a. Nature of agent  
   b. Concentration of the agent  
   c. Quantity of the agent  
   d. Length of time the agent is in contact with tissue  
   e. Degree of tissue penetration  
   f. Mechanism of action  
B. Diagnosis  
1. Chemical burns are deeper than initially appear and may progress with time  
   a. Fluid resuscitation needs often underestimated  
   b. Watch for renal/liver/pulmonary damage  
C. Treatment  
1. Initial treatment is dilution of the chemical with water  
2. Special attention to eyes — after copious irrigation with saline, consult ophthalmologist  
3. After 12 hours initial dilution, local care of the wound with debridement, topical antibiotics, and eventual wound closure is same as for thermal burn
D. Of particular note are:
1. Gasoline
   a. Excretion by lung
   b. May cause large skin burn, if immersed
   c. Watch for atelectasis, pulmonary infiltrates; surfactant is inhibited
2. Phenol
   a. Dull gray color to skin, may turn black
   b. Urine may appear smoky in color
   c. Spray water on burn surface
   d. Wipe with polyethylene glycol
   e. Direct renal toxicity
3. Hydrofluoric acid
   a. Irrigate copiously with water
   b. Subcutaneous injections of 10% of calcium gluconate
   c. Monitor EKG patients — may become hypocalcemic
   d. Pulmonary edema may occur if subjected to fumes
4. White phosphorous
   a. Do not allow to desiccate — may ignite
   b. Each particle must be removed mechanically
   c. Copper sulfate (2%) may counteract to make phosphorous more visible (turns black in color)
   d. Watch for EKG changes (Q – T interval and S – T and T wave changes)
   e. May cause hemoglobinuria and renal failure

III. ELECTRICAL INJURIES
   A. Pathophysiology
      1. Effects of passage of electric current through the body depend on:
         a. Type of circuit
         b. Voltage of circuit
         c. Resistance offered by body
         d. Amperage of current flowing through tissue
         e. Pathway of current through the body
         f. Duration of contact
      2. Tissue resistance to electrical current increases from nerve (least resistant) to vessel to muscle to skin to tendon to fat to bone

B. Diagnosis
   1. Types of injury
      a. Arc injury: localized injury caused by intense heat
      b. Injury due to current
         i. Due to heat generated as current flows through tissue
            (a) Injury more severe in tissue with high resistance (i.e. bone)
            (b) Vessels thrombose as current passes rapidly along them
         ii. Effects of current may not be immediately seen
   2. Cardiopulmonary
      a. Anoxia and ventricular fibrillation may cause immediate death
      b. Early and delayed rhythm abnormalities can occur
      c. EKG changes may occur some time after the burn
   3. Renal
      a. High risk of renal failure due to hemoglobin and myoglobin deposits in renal tubules
         i. Requires higher urine flow (75cc/hr in adults)
         ii. Must alkalinize urine to keep hemoglobin and myoglobin in more soluble state
         iii. Mannitol may be useful to clear heavy protein load
   4. White phosphorous
      a. Do not allow to desiccate — may ignite
      b. Each particle must be removed mechanically
      c. Copper sulfate (2%) may counteract to make phosphorous more visible (turns black in color)
      d. Watch for EKG changes (Q – T interval and S – T and T wave changes)
      e. May cause hemoglobinuria and renal failure
6. Vascular effects
   a. Vessel thrombosis progresses with time
   b. Delayed rupture of major vessels can occur
7. Cataract formation — late complication
8. Seizures
D. Treatment
1. CPR if necessary
2. Fluids — usually large amounts
   a. No formula is accurate because injury is more extensive than can be predicted by skin damage
   b. Alkalinate with NaHCO₃ if myoglobinuria or hemoglobinuria present
3. Monitoring
   a. CVP or pulmonary wedge pressure helpful since total capillary leak does not occur as it does in a thermal burn
   b. Maintain urine output at 75-100cc/hr until all myoglobin and/or hemoglobin disappears from urine
4. Wound Management
   a. Topical agent with good penetrating ability is needed [i.e. silver sulfadiazine (Silvadene®) or mafenide acetate (Sulfamylon®)]
   b. Debride non-viable tissue early and repeat as necessary (every 48 hrs) to prevent sepsis
   c. Major amputations frequently required
   d. Technicium-99 stannous pyrophosphate scintigraphy may be useful to evaluate muscle damage
5. Treat associated injuries (e.g. fractures)

IV. COLD INJURIES
The two conditions of thermal injury due to cold are local injury (frostbite) and systemic injury (hypothermia)
A. Frostbite
1. Pathophysiology
   a. Formation of ice crystals in tissue fluid
      i. Usually in areas which lose heat rapidly (e.g. extremities)
   b. Anything which increases heat loss from the body such as wind velocity, or decreases tissue perfusion, such as tight clothing, predisposes patient to frostbite
   c. Ability of various tissue to withstand cold injury is inversely proportional to their water content
2. Treatment
   a. The key to successful treatment is rapidrewarming in a 40˚C waterbath
      i. Admission to hospital usually required
         (a) Tetanus prophylaxis
         (b) Wound management
         (c) Physical therapy
            (i) Maintenance of range of motion important
            (ii) Daily whirlpool and exercise
         (d) Sympathectomy, anti-coagulants, and early amputation of questionable value in controlled studies
   ii. Usually wait until complete demarcation before proceeding with amputations. Non-viable portions of extremities will oftenautoamputate with good cosmetic and functional results.
B. Hypothermia
1. Diagnosis
   a. Core temperature < 34˚C
   b. Symptoms and signs mimic many other diseases
   c. High level of suspicion necessary during cold injury season
2. Treatment
   a. Must be rapid to prevent death
   b. Monitor EKG, CVP, and arterial blood gases and pH during warming and resuscitation, maintain urine output of 50cc/hr
   c. Begin Ringer’s Lactate with 1 ampule NaHCO₃
   d. Oral airway or endotracheal tube if necessary
   e. Rapidly rewarmin 40˚ hydrotherapy tank (requires 1-2 hours to maintain body temperature at 37˚C)
   f. Treat arrhythmias with IV Lidocaine drip if necessary
   g. Evaluate and treat any accompanying disease states
V. LIGHTNING INJURIES
   A. Cutaneous effects — lightning strikes may cause cutaneous burn wounds
      1. Contact burns from clothing on fire or contact with hot metal (i.e. zippers)
      2. Entry and exit burns are usually small, may be partial or full thickness
      3. Lightning burns are not the same as electrical burns — don’t get deep tissue injury
   B. May have temporary ischemic effects on extremity — pallor or neurologic deficits. Spontaneous recovery after a few hours is the rule — probably due to local vasoconstriction
   C. Systemic effects can occur such as arrhythmias, cataracts, CNS symptoms

CHAPTER 8 — BIBLIOGRAPHY

THERMAL INJURIES