Overview of Burn Injuries

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- 2. Etiology and Epidemiology of Burns
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- 4. Pathophysiology of Burn Injury
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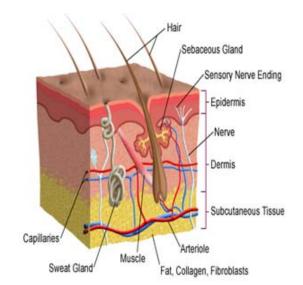
Normal Skin Anatomy

Skin is the largest "organ" of the body; it constitutes about 17% of the total body weight. The importance of skin is frequently overlooked. The epidermis provides a water-proofing and bacteria-proofing layer, whereas the dermis gives the skin its toughness and durability.

The skin is made up of the following layers

Epidermis The epidermis is the thin outer layer of the skin which consists of the following three parts:

- Stratum corneum (horny layer): This layer consists of fully mature keratinocytes which contain fibrous proteins (keratins). The outermost layer is continuously shed.
- Keratinocytes (squamous cells) layer: This
 layer contains living keratinocytes (squamous
 cells), which mature and form the stratum
 corneum.



Basal layer: The basal layer is the deepest layer of the epidermis, containing basal cells
which continually divide, forming new keratinocytes, replacing the old ones that are shed
from the skin's surface. The epidermis also contains melanocytes, and immunologic
Langerhans' cells.

Dermis: The dermis is held together by a protein called collagen, made by fibroblasts. This layer also contains blood and lymph vessels, hair follicles, sweat glands, and cutaneous nerve receptors.

Subcutaneous fat layer: This is the deepest layer of skin. The Subcutaneous layer "subcutis" consists of a network of collagen and fat cells, helps conserve the body's heat and protects the body from injury by acting as a "shock absorber."

Functions of the Skin

- Identification
- Cosmetic Appearance
- Production of Vitamin D
- Sensations of touch, pain and temperature
- Maintenance of Body Temperature
- A protective Barrier
 - Protection from environment UV radiation
 - Prevents evaporative water loss
 - Protection from microorganisms

Etiology and Epidemiology of Burns

Burns is a coagulative necrosis, commonly affecting the skin and subcutaneous tissue, caused by insult by thermal, electrical, chemical, or electromagnetic energy. Burn injury usually results from transfer or generation of "thermal" energy to the body. There are many types of burns caused by thermal, radiation, chemical, or electrical contact.

Thermal burns - burns due to external heat sources which raise the temperature of the skin and tissues and cause tissue and cell death. Hot surfaces, scalding liquids, steam, and flames, when coming in contact with the skin, can cause thermal burns.

Electrical burns – progressive tissue damage due to passage of electric current (high of low voltage) with the living body.

Radiation burns - burns due to prolonged exposure to ultraviolet rays of the sun, or to other sources of radiation such as x-ray.

Chemical burns - burns due to strong acids, alkalis, detergents, or solvents coming into contact with the skin and/or eyes.

Key points

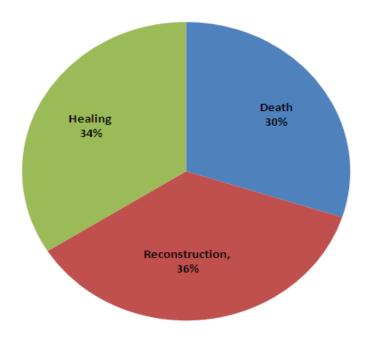
- Burns are a major cause of injury and death worldwide.
- Flame burns are the most common type.
- 90% of burns are preventable
- Young children, elderly, and mentally or physically compromised are at particular risk.
- Death is more likely with increasing age, increasing burn size, and presence of inhalational injury.

Frequency and incidence

The highest incidence of serious burn injury occurs in young adults (20-29 y) followed by children younger than 9 years. Serious burn injuries occur most commonly in males (67%). For children younger than 2 years, liquid scalds and hot surface burns account for nearly all serious burn injuries. After age 2 years, flame burn is the most common cause of serious burn injuries, accounting for nearly one third of all serious burns.

Burn statistics in Egypt

- 0.1% of population is affected by major burns.
- Burn is the 2nd leading cause of accidental death (after MVA).
- The average hospital stay is 45 days.
- Domestic causes account for 75% of burns.
- The kitchen is the most frequent area, the second area is the bathroom
- Children are the majority of admitted patients.
- Complications are greater after the age of 60.
- About one third of cases (34%) heals spontaneously while, Death rate is 30%, and more than a third (about 36%) of cases need later reconstruction.



Assessment of Burn Injury

Burn injuries cause a disease that severely affects the whole organism. The problems of prognosis and treatment depend on the extent and depth of the wounds, as also on the localization of the burn. The localization of the burn is also important. Particularly dangerous are burns of the face and throat area because of the high risk of injury to the respiratory tract, resulting in its closure due to laryngeal edema. There is also the risk of poisoning by carbon monoxide and - within closed space. Also crucial are the age of the patient and any attending diseases.

Assessment of Burn Size

Burn injuries cause a disease that severely affects the whole organism. The problems of prognosis and treatment depend on the extent and depth of the wounds, as also on the localization of the burn. Also crucial are the age of the patient and any attending diseases. The amount of body surface burned has a direct effect upon the outcome. Any patient with more than 50% of his Body Surface Area (BSA) burned is considered to have an extensive burn.

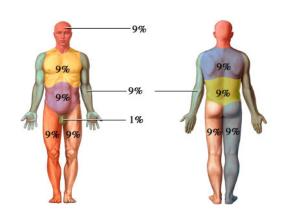
Methods to evaluate burn size: Palm Method

A quick method to evaluate scattered or localized burns. The patient's own palm = 1 % TBSA



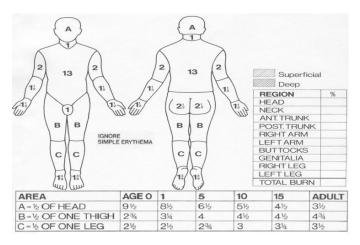
Rule of Nines

Rule of Nines which indicates the percentage of total body area accounted for by various parts of the body; 9% for each of the arms & head, 18% for each of the lower limbs, front of trunk and back of the trunk and 1% for the perineum. The palmar surface of the hands accounts for 1%. Simple erythema should not be counted.



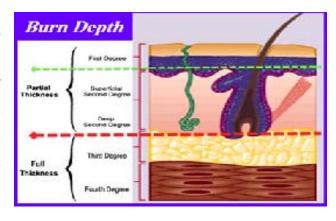
Lund & Browder chart

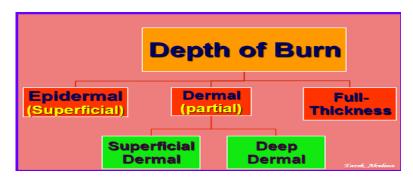
For a more accurate estimation, especially in children whose proportions are different from adult one; a **Lund & Browder chart** should be used which makes a more accurate allowance for the differences in body surface area with age and for the differences in burn depth inside the affected area(s).



Assessment of Burn Depth

Burn depth has an impact on healing time, the need for hospitalization and surgical intervention, and the potential for scar development. Although accurate classification is not always possible initially, the causes and physical characteristics of burns are helpful in categorizing their depth.





The traditional classification of burns as first, second or third degree is being replaced by the designations of superficial, superficial partial thickness, deep partial thickness and full thickness.

Superficial burns

- Only the epidermis is affected.
- Redness, tenderness, and pain are the hallmarks.
- Pin brick sensations intact
- Never blisters with no metabolic response
- Not calculated in burn extent
- Regeneration is rapid and complete without scarring
- Example: Sunburns



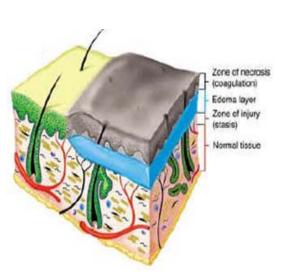
Superficial partial-thickness burn

The burn involves the epidermis and superficial dermis. Blistering wounds that blanch with pressure are characteristic of superficial partial-thickness burns. These wounds are also typically moist and weeping.

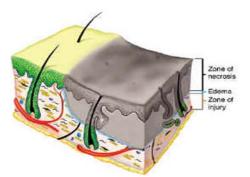
- The burns appear pink, moist, and soft and tender.
- Thin-walled, fluid-filled blisters are usually found.
- They heal within 7-14 days after injury, usually without scarring, by outgrowth of epithelial buds from the viable pilosebaceous units and sweat glands.







Deep partial-thickness burn



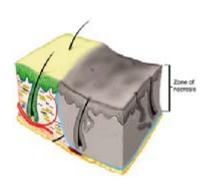
The deeper "reticular" dermis is burned. These burns are typified by easily unroofed blisters that have a waxy appearance and they do not blanch with pressure.

Characteristics:

- The burn extends into the reticular dermis.
- Skin color is usually a mixture of red and blanched white, and capillary refill is slow.
- Blisters are **thick-walled** and commonly ruptured.
- Two-point discrimination may be diminished, but pressure and pinprick applied to the burned skin can be felt.
- Tissue may undergo spontaneous epithelialization from the few viable epithelial appendages at this deepest layer of dermis and heal within 3-6 weeks (if no infection arises).
- Greater potential for hypertrophic scar formation exists.
- Contraction across joints, with resulting limitation in range of motion, is a common sequela.

Full-thickness burns

Differentiating a deep partial-thickness burn from a full-thickness burn can be quite difficult initially. **Repeated evaluation** and revisions of burn-depth estimations are often necessary in the first 24 to 72 hours and may be required through the first two or three weeks. It is also possible to have a full-thickness burn underneath a blister, which is usually a

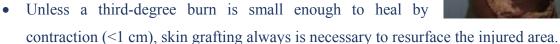


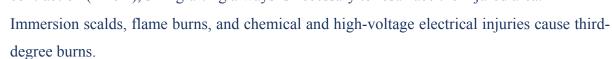
characteristic feature of a partial-thickness burn. Furthermore, thin skin sustains deeper burn injuries than may be suggested by the initial appearance of the wound. Thin skin is common on

the volar surface of the arms and on the medial thigh, perineum and ears. All skin can be presumed to be thin in children < 5 years and in adults > 55 years.

Characteristics:

- Third-degree burns are full-thickness burns that destroy both epidermis and dermis.
- The capillary network of the dermis is completely destroyed.
- Burned skin has a white or leathery appearance with underlying clotted vessels and is anesthetic.





Burn depth summary: cause, diagnosis and healing

Degree		CAUSE	APPEARANCE	PAIN	HEALING	SCAR
SUPERFICIAL (Epidermal)		Sunburn, Radiation	Red, painful, no blisters	Moderate -to - severe	7-10 days	No scarring
Partial (DERMAL)	SUPERFICIAL Dermal	hot liquid, flash flame	wet, pink, + blisters	severe	10-20 days	minimal
	DEEP Dermal	chemicals, direct contact flames	Wet-dry, pink-white ± thick blisters	minimal	2-6 weeks better grafted	moderate or severe
FULL- THICKNESS		chemicals, flames, Explosion, with very high temp.	dry, white Leathery, or char	none	need graft	Mild - severe depending on timing and type of graft

Remember: there is no sharp border to separate different depths and in the same burn victim you may find a complex combination of all three types, moreover the depth may change with time, especially if infection occurs.



The pathophysiology of the burn injury

Burn is a prolonged process, unlike many other forms of trauma. Burn insult to the skin leads to the release of numerous factors (histamine and histamine-like substances). These mediators increase capillary permeability and induce a hypermetabolic state. Proteins and fluids pour out into the damaged area. If the wound is more than 30% of the body surface area, there is an appreciable loss of fluid into the undamaged tissues. Thermal damage to the skin leads to the release of numerous factors which increase capillary permeability. Proteins and fluids pour out into the damaged area and, roughly speaking, protein and fluid losses reflect the area of the injured skin. If the wound is large enough, more than 30% of the body surface area, there is an appreciable loss of fluid into the undamaged tissues. Change in the capillary permeability is maximal at about 8 h after the injury and returns towards normal over the next 36 hours1. Water and electrolytes diffuse back into the circulation leaving the proteins trapped in the interstitial spaces where they are eventually removed by the lymphatics. There are multiple system disturbances including Metabolic, Vascular, Gastrointestinal and Immune system disturbances.

• Metabolic disturbances

o Increase in core body temperature → Hypermetabolic state with Increased oxygen and calorie requirements

• Vascular Changes

Fluid Shift during the first 36 hours after burn

- Local Vasodilatation adjacent to burn injury → ↑ capillary hydrostatic pressure and ↑ capillary permeability with continuous leak of plasma from intravascular space into interstitial space
- Hemoconcentration with electrolytes and acid-base disturbances.

Fluid Remobilization after 36 hours

- Capillary leak ceases and fluid shifts back into the circulation
- Restores fluid balance and renal perfusion → diuresis
- Hemodilution with associated Hyponatremia and Hypokalemia

• Gastrointestinal (G I) disturbances

- o Decreased or absent G I motility (may need NG tube)
- o Curling's ulcer formation

Immunologic disturbances

- Loss of protective barrier
- o Increased risk of infection
- o Suppression of humoral and cell-mediated immune responses.

Pathology of burn injury

The classic description of the burn wound and surrounding tissues is a system of several circumferential zones radiating from primarily burned tissues, as follows:

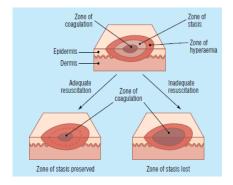
- 1. Zone of coagulation A nonviable area of tissue at the epicenter of the burn
- 2. Zone of ischemia or stasis Surrounding tissues (both deep and peripheral) to the coagulated areas, which are not devitalized initially

but, due to microvascular insult, can progress irreversibly to necrosis over several days if not

resuscitated properly

3. Zone of hyperemia - Peripheral tissues that undergo vasodilatory changes due to neighboring inflammatory mediator release but are not injured thermally and remain viable





NOTE: The aim of the resuscitation is to support the circulation in the zone of stasis to prevent its conversion into necrosis.

Treatment of Burns

First Aids

Cool tap water (NO ICE) should be run or sponged over the burnt area for 30 minutes. It will not be of value if commenced greater than 3 hours post burn. The beneficial effects of immediate cold water treatment of burned skin appear to be related to three factors:

- 1. Cold also inhibits burn wound histamine release, which in turn blocks local and remote histamine-mediated increases in vascular permeability. This minimizes edema formation and intravascular volume losses.
- 2. Cold suppresses the production of thromboxane, implicated as the mediator of vascular occlusion and progressive dermal ischemia after burn injury.

Note: Don't Use Iced water as it may induce intense vasoconstriction and burn progression.

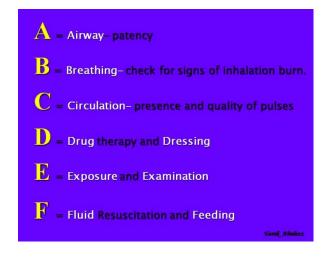
Referral Criteria of Burn Victim

According to the American burn Association Burn Center Referral Criteria:

- Dermal Burns involving more than 10% BSA in patients < 10 or > 50 years of age
- Dermal Burns involving more than 20% BSA in any age
- Burns of special areas: Face, Hand, Feet, Buttocks, Perineum, genitalia and Major joints
- Full-Thickness Burns of more than 5% BSA
- Special Burns: Electric burns, Chemical burns "when serious", Eye burns and Inhalation injury
- Burns with a concomitant mechanical injury or pre-existing medical illness

Essential ER Management Points:

- REMOVE Clothes: Clothing can retain heat, even in a scald burn.
- Secure wide bore IV access (Multiple)
- Determine the percentage area of burn (Rule of 9's)
- ABCDE



Fluid Resuscitation of burned patient:

Adequate fluid management is critical for survival from a major burn injury. Knowledge of fluid management following a major burn is very important. All the resuscitation fluid formulas are guidelines. Their success relies on adjusting the amount of fluid against patients' monitored physiological parameters. The amount of fluid depends on severity of the injury, age, physiological status and any associated injury. Because the greatest plasma loss occurs in the first 24 hours post burn, the greatest replacement needs to occur in that period. In optimising fluid resuscitation, the amount of fluid should be just enough to maintain vital organ function without producing pathological changes.

The most commonly used Resuscitation Formulas

Evan's formula

- For the first 24 hours after burn, normal saline at 1 ml/kg/% TBSA burn + colloid at 1 ml/kg/% TBSA burn+ D5W (dextrose 5% in water) 2000 ml.
- For second 24 hours, give half of first 24 hour requirements + the same amount of glucose in water as in the first 24 h

Parkland Formula – Parkland's formula doesn't use colloids in the first day and uses a higher rate of infusion.

- The total volume of the first 24 hours of resuscitation (with Ringer lactate solution) is approximately 4 mL/kg body weight X percentage burn. Half the volume is given in the first 8 hours postburn, with the remaining volume delivered over 16 hours.
- For the second 24 h: colloid at 20–60% of calculated plasma volume is administred to maintain adequate urinary output

Ringer Lactate solution is a relatively isotonic crystalloid solution that is the key component of almost all resuscitative strategies. It is preferable to normal saline for large-volume resuscitations because its lower sodium concentration and higher pH concentration are closer to physiologic levels.

Multiple formulas exist with variations in both the volumes per weight suggested and the type or types of crystalloid or crystalloid-colloid combinations administered. To date, no single recommendation has been distinguished as the most successful approach.

Calculated volumes from all of the formulas should be viewed as **guidelines**. Blind adherence to a derived number can lead to significant over-resuscitation or under-resuscitation if not interpreted within the clinical context.

Albumin is the plasma protein that most contributes to intravascular oncotic pressure. When administered intravenously as a 5% solution, approximately half the volume remains intravascularly, as opposed to 20-30% of crystalloid solutions. Alternatively, some centers prefer using **fresh frozen plasma** over using albumin because of the theoretic advantage of replacing the whole range of plasma proteins that are lost rather than just the albumin fraction.

Hypertonic saline solutions, ranging in concentration from 180-300 mEq sodium per liter, have many theoretic benefits like: reduction in volume requirements by mobilizing intracellular fluid into the vascular space by the increased osmotic gradient. Hypertonic saline is most beneficial for those patients with the most limited cardiopulmonary reserves, those with inhalation injury, and those with larger burns approaching 40% or more. Close monitoring of serum sodium levels is mandatory.

Monitoring of resuscitative adequacy

- Regular check-up of vital signs
 - 1. General state restlessness suggests hypoxia.
 - 2. Warm peripheries.
 - 3. Pulse under 100 per min
- The urine output should be 0.5-1 ml/kg body weight/hour
- Monitoring of Central Venous Pressure (CVP)
- 5 Daily urea, electrolytes, hemoglobin and hematocrit levels.

Nutrition and Burn

Metabolic demands of the thermally injured patient have been evaluated, and have been found that most major burns require at least double of the normal daily requirement. Therefore, twice the basal energy expenditure is a generous estimate of the total caloric requirement in burn patients. Burn patient should ne given a variety of foods that have good nutrients to help the skin heal. Vitamins A and C are important vitamins for the skin. Some foods that have Vitamin A and C are oranges, grapefruits, tomatoes, strawberries, broccoli, and carrots. Many enriched cereals also contain vitamins. Foods that contain protein such as meat, fish, eggs, peanut butter, chicken, and milk are also important to skin healing.

Drug Therapy for Burn Victim

■ **Pain Control**: Once vital signs have been stabilized, intravenous opiate analgesia (morphine) is administered to the patient to combat neurogenic shock and to help for better examination and assessment of the burn and to facilitate wound dressing.

IV is used due to:

- GI function is slowed or impaired because of shock and/or paralytic ileus
- I.M. injections will not be absorbed well (sluggish circulation)
- **Beta Blockers**: Patients with burns > 40% are always catabolic, and their metabolic derangement persists for at least a year after injury. Propranolol reduces hypermetabolism, heart rate, oxygen demand and resting energy expenditure. Betablockers decrease mortality, wound infection rate, and wound healing time.
- Vitamin and mineral Supplementation: High dose vitamin C given I.V. infusion in the first 24 hours after burn has been documented to ameliorate burn inflammatory process, promote healing and decrease in the required fluid resuscitation. Other antioxidants like Vit E, Zink and vit A are recommended
- **Peptic ulcer Protector:** Proton-Pump Inhibitor (Omeperazole) or an H2 blocker (cimetidine, ranitidine) is given IV to protect against peptic ulcer formation
- **?? Tetanus:** Tetanus immunization may be given because of the likelihood of anaerobic burn-wound contamination. However, I have never given it for more than 20 years of practice without any recorded cases of tetanus.
- Antimicrobial agents: Systemic antibiotics are required when there is Fever or septicemia. Carefully assess wounds for signs and symptoms of infection, including erythema, edema, increased heat, and discomfort. Obtain culture specimens if you suspect infection. Monitor the patient's vital signs and temperature every 4 hours. When infection is suspected, the physician may prescribe I.V. antibiotics. The use of prophylactic antibiotics (against gram-positive in the first 3 days.....then anti-Pseudomoneal) is debatable.

Burn Wound Management

Initial treatment of the burn wound involves cleansing the wound with Saline then Betadine solution. Ruptured blisters are removed with scissors. After wound cleansing, cover with a topical antimicrobial dressing. Topical antibiotics decrease microbial growth and reduce invasive infection. Prophylactic systemic antibiotics are not recommended because they do not prevent wound sepsis. Systemic antibiotics may be indicated when cellulitis is evident in surrounding unburned tissue.

Dressings: Repeated dressings until complete healing occurs or surgical intervention is required for wound closure. The wound is cleansed with soft sponges soaked in diluted Betadine solution, to completely remove any debris, dead tissue (eschar) or previously applied tpical agent. After wound cleansing, cover the wound with antibiotic-impregnated Vaseline-gauze dressing. The ideal burn dressing shouldcControl colonisation, promote rapid granulation and epithelialisation, be non-adherent in order to minimise pain and prevent damage to granulating tissue and regenerating epithelium at dressing change, does'nt hinder movement or compliance with physiotherapy and doesn't compromise the circulation.

Dressing of Burned hand: Due to the functional importance of the hand and the high risk of contractures associated with burns, it is essential that the hand and fingers are mobilised throughout the treatment period. In minor injuries each finger should be dressed individually without restricting movement. Hand exercises should be encouraged. In larger superficial burns to the hand - for example, scalds - the entire hand may be placed in a polythene bag for 24-48 hours.

Management of burn blisters: Burn blister management is open to debate. This is because burn blister fluid can have beneficial and detrimental effects. There are two schools of thought on the de-roofing of blisters. Some practitioners believe that they should be completely de-roofed, as any remaining skin may be a potential source of infection. Others believe that, once the blister has burst, the sac should be retained and allowed to act as a biological dressing.

Escharotomy: Full-thickness circumferential burn of an extremity can result in vascular compromise. Loss of Doppler ultrasound signals in peripheral arteries is an indication for escharotomies of the burned extremity. A period of 3-8 hours is required for edema to develop sufficiently to increase tissue pressure. When tissue compartment pressures are greater than 40 mm Hg, escharotomies of the full-thickness burn prevent this ischemic injury. Note that the most common cause of absent pulses in an extremity is hypovolemia with peripheral vasoconstriction, not increased interstitial pressure.

Surgical debridement: The standard of care for full-thickness burns is burn wound excision and grafting. The mortality of patients with massive burns is reduced by early tangential excision of the entire wound, followed by skin closure with an autograft from unburned areas on the patient or an homograft from a skin donor.

Grafting: Indications for graft surgery are full-thickness burns or partial-thickness burns that are unlikely to heal within 3 weeks. If the burn fails to heal in 3 weeks, the risk for hypertrophic scar and contracture formation increases and the healed wound exhibits an aesthetically displeasing scar.

Special Burns

Inhalation injury

When this accompanies thermal trauma, it increases the magnitude of total body injury and requires increased volumes of fluid and sodium to achieve resuscitation from early burn shock (Navar, 1985). These patients often require 40–50% more fluid, whatever resuscitation regimen is used (Yowler and Fratianne, 2000). However, due to this increase patients are at risk of developing pulmonary oedema. Reducing the fluid to avoid this may result in inadequate cardiac output and lung perfusion, which can impair ventilation perfusion.

First you assess her airway, breathing, and circulation. Look for signs of a compromised airway and inhalation injury, which can be fatal. Someone confined or trapped in an enclosed space during a fire or involved in an explosion is at risk for developing pulmonary complications from inhaling toxic fumes and smoke.

Signs and symptoms that alert you to possible inhalation injury include:

burns to the face, head, ears, neck, or chest

singed facial hair, nostrils, or lips

blistering on the face or mouth

irritated or tearing eyes

carbonaceous (sooty) sputum

dyspnea or tachypnea

Hoarse voice

Wheezing, persistent cough

Electrical Burns

All electrical burns are full-thickness. Electrical burns are caused by electricity as it passes through the body and meets resistance from body tissue. The heat it causes is proportional to the amperage of the current and the electrical resistance of the body. An electrical injury occurs when electricity is converted into heat as it travels through tissue. Approximately 15% of these patients have associated multiple trauma. External burns are usually caused at the entry and exit sites, but the cutaneous injury usually does not reflect the presence of deep tissue damage. Nerves, blood vessels, and muscles are less resistant and more easily damaged than fat or bone. Organs such as the brain, heart, and lungs are damaged quickly and easily. The smaller the body part the more intense the heat. Considerable damage can occur in the extremities. Muscle damage should be suspected if the urine is grossly pigmented (light pink to deep brown). Electrical current can cause the heart to fibrillate. Alternating current (AC) can cause titanic muscle contractions and intensify the patient's grip on the electrical source, increasing the time of exposure.

High Tension Electrical Burns

Therapeutic guidelines in the treatment of electrical injuries are similar to thermal injuries, but some points differ. Because of the deep-tissue damage, resuscitation volumes calculated from the visible injury may substantially underestimate actual needs to gother with

careful monitoring. Serial neurovascular monitoring assumes is mandatory. With any evidence of increasing neurovascular compromise, both escharotomy and fasciotomy of the affected part must be done without hesitation to avoid compartment syndromes and ischemic tissue loss.

The presence of pigment in the urine is an indication of deep-muscle damage. It is essential that a diuresis of 100 cc urine per hour in adults and 1.5–2 ml/kg/hr in the child is maintained. Optimal management includes fluid loading to





initiate diuresis and osmotic diuretics (e.g., mannitol), if necessary. Alkalinization of the urine with intravenous sodium bicarbonate will assist in preventing myologlobin precipitation. Operative debridement is the most important part of therapy for electrical injuries. In some cases of high-voltage injuries, we may need to perform amputation of a limb.

Low Tension Oral Burns

Low-voltage electricity is the leading cause of electrical injury in children, especially 1 to 2 years old. Sucking an extension cord is responsible for most of the injuries followed by biting on an electric cord. The local mouth burn is characteristically grayish-white in color and indented at the center due to tissue necrosis. The oral burn may involve the lip, tongue, or oral mucosa and underlying mandibular bone. The most frequent site is the lip, in particular the commissural area between upper and lower lips. The edema of the lips may be intense, impairing

control of saliva. Edema subsides over the next 5 to 10 days and local necrotic tissue begins to slough. Bleeding from the labial artery is a common occurrence during the period of slough (7 to 21 days) and should be anticipated. Granulation tissue then develops, followed by collagen deposition and wound remodeling. Local adhesions and microstomia may develop over a period of 3 to 5 months. Injury to the underlying bone will result in dental abnormalities over time.



Treatment

Initial hospitalization is recommended to treat the local burn and observe for any current related injuries. Tetanus prophylaxis is necessary, but systemic antibiotics do not appear to be particularly beneficial. Local wound care with gentle washing followed three to four times daily by local application of a petroleum-based antibiotic ointment.

Chemical Burns

Common strong acids and alkali used in industry cause the majority of injuries. The burn injury is typically caused by coagulation necrosis of tissue rather than by direct heat production. The degree of tissue injury is dependent on the toxicity of the chemical and the duration of exposure. The burn wound is characteristically gray to brown in color due to the chemically

denatured protein. Persistent burning pain is commonly described as the burning in process continuous as long as the chemical is in contact with the skin. Burns are invariably deeper than first appearance indicating ongoing injury. Also the degree of tissue damage takes longer to declare itself such that after 13 to 24 hours the wound is invariably deeper.

General Treatment

- Removal of clothing contacting the skin
- Immediate copious irrigation with tepid water
- Identify the chemical for specific therapy
- Monitor for systematic toxicity
- Monitor body Temperature avoid hypothermia

Acids: Acids release hydrogen ions and reduce pH from a neutral 7 down to values as low as 0. Hydrogen ions will catalyze protein hydrolysis into amino acids. Other effects as seen in concentrated sulfuric acid injuries include heat generation and desiccation, producing a mixed injury. Tungstic, picric, sulfosalicylic, tannic, trichloacetic, and cresylic acids are all strong protoplasmic poisons which form hard eschars. These acids can be absorbed producing renal and hepatic injury.



Alkali: Multiple chemical agents are involved in caustic alkali burns. Lime, potassium hydroxide, and sodium hydroxide are the most common agents causing chemical injury. The mechanism by which these alkali injuries are caused is due to three factors:

- Saponification of fat causes fatty tissue to lose its function with increased heat reaction.
- Extraction of water from cells causing desiccation
- Bind with the proteins of the tissues to form alkaline proteinates.

The extent of the damage caused by an alkali substance depends on its concentration, amount, and time of contact with the skin. Alkalis are capable of deep penetration, and can cause severe pain. For treatment, it is necessary to remove the causative substance as quickly as possible by washing with large volumes of water. The initial treatment for burns caused by strong alkaline solution is copious irrigation with water.

Ocular damage is a common sequel to alkali injury. Damage is related to the pH: the higher the pH, the greater the damage to the eye. Such damage may be made worse by repeated attempts to wipe off the chemical agent with the hands or inadequately washing the eyes. The policy is to treat with copious irrigation then induction of cycloplegia and topical anesthetics as soon as possible.

Cement: Cement is an alkali, but warrants special mention. Cement (calcium oxide) penetrates clothing and reacts with sweat, and an exothermic reaction takes place releasing heat. The dry powder will cause a desiccation injury if not exposed to moisture. Hydrated calcium oxide becomes calcium hydroxide.



Treatment consists of removal of contaminated clothing and washing the burn area with soap and water and then the area is dried thoroughly.

Complications of burns

Hypertrophic scars, Keloids and Contractures

Predisposing factors

- Young age
- Dark races
- Sites of skin tension e.g. over the sternum, Ear lobule, shoulders and deltoid region.
- Healing by secondary intention: Infection, trauma or sepsis

How to prevent?

1-Proper diagnosis of burn depth and early grafting of deep burns

2-Pressure Garments:

The application of external constant pressure by pressure garments keeps the scars flat and pliable.

3-Creaming and massage

Newly healed or grafted skin in thin and has no oil glands. Creaming and massage play a vital role in maintaining the lubrication and elasticity of skin and therefore assisting in maintaining the range of movement.

4-Protection from sun light

In the first year after burn injury healed skin is more prone to sunburn and hyperpigmentation and prolonged exposure to sun light should be avoided and the a powerful sun blocking cream (with high Sun Protection Factor SPF) is used.

5- Silicone Gel

It can be used in the prevention and treatment of hypertrophic scarring, causing the scar to soften and flatten without the use of pressure.

6-Management of itching:

- Avoid scratching keep fingernails trimmed
- Good personal hygiene Massage- Keep skin well moisturized
- Avoid over heating –Wear cotton clothes and avoid synthetic clothes
- Pressure garments –
- Topical application of Antihistaminic and/or Corticosteroids creams

7-Splintage (Static or dynamic) in position of function in cases of burns around joints