

Burns: Epidemiology, Pathophysiology and Prevention

Introduction

- A burn is an injury to the skin or other organic tissue primarily caused by heat or due to radiation, radioactivity, electricity, friction or contact with chemicals.
- Thermal (heat) burns occur when some or all of the cells in the skin or other tissues are destroyed by:
 - hot liquids (scalds)
 - hot solids (contact burns), or
 - flames (flame burns).

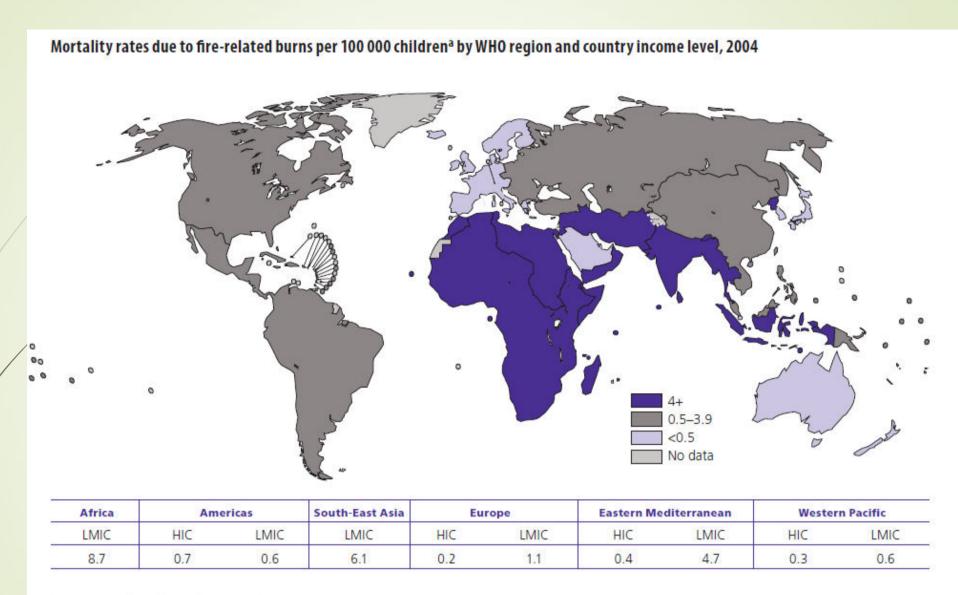


Epidemiology of Burns

The global scenario

- Burns are a global public health problem, accounting for an estimated 265 000 deaths annually. The majority of these occur in low- and middle-income countries and almost half occur in the WHO South-East Asia Region.
- Non-fatal burns are a leading cause of morbidity, including prolonged hospitalization, disfigurement and disability, often with resulting stigma and rejection.
- Burns are among the leading causes of disability-adjusted life-years (DALYs) lost in lowand middle-income countries.
- In 2004, nearly 11 million people worldwide were burned severely enough to require medical attention.





These data refer to those under 20 years of age.

HIC = High-income countries; LMIC = low-income and middle-income countries.

Source: WHO (2008), Global Burden of Disease: 2004 update.

India





Who is at risk?

Gender

Females and males have broadly similar rates for burns, according to the most recent data. (In contrast to other injuries where males have a higher rate)

Age

children are particularly vulnerable to burns. Burns are the 11th leading cause of death of children aged 1–9 years and are also the fifth most common cause of non-fatal childhood injuries.

Regional factors

- There are important regional differences in burn rates.
- Children under 5 in the WHO African Region have almost 3 times the incidence of burn deaths than infants worldwide

Socioeconomic factors

- People living in low- and middle-income countries are at higher risk for burns than people living in high-income countries.
- Within all countries however, burn risk correlates with socioeconomic status.

Other risk factors

- occupations that increase exposure to fire;
- poverty, overcrowding and lack of proper safety measures;
- placement of young girls in household roles such as cooking and care of small children;
- underlying medical conditions, including epilepsy, peripheral neuropathy, and physical and cognitive disabilities;
- alcohol abuse and smoking;
- easy access to chemicals used for assault (such as in acid violence attacks);
- use of kerosene (paraffin) as a fuel source for non-electric domestic appliances;
- inadequate safety measures for liquefied petroleum gas and electricity.



- In which settings do burns occur?
- Burns occur mainly in the home and workplace.
- Community surveys in Bangladesh and Ethiopia show that 80–90% of burns occur at home.
- Children and women are usually burned in domestic kitchens, from upset receptacles containing hot liquids or flames, or from cookstove explosions.
- Men are most likely to be burned in the workplace due to fire, scalds, chemical and electrical burns.

Classification of Burn Injuries



By mechanism of cause

- 1. thermal
- 2. Electrical
- 3. Chemical
- 4. Inhalational
- result of breathing in superheated gases, steam, hot liquids or noxious products of incomplete combustion.
- cause thermal or chemical injury to the airways and lungs.

Thermal Burns

Thermal burns involve the skin and may present as: –

- a) scalds hot liquid or steam
- b) contact burns hot solids or items such as hot pressing irons and cooking utensils, as well as lighted cigarettes
- c) flame burns flames or incandescent fi res, such as those started by lighted cigarettes, candles, lamps or stoves

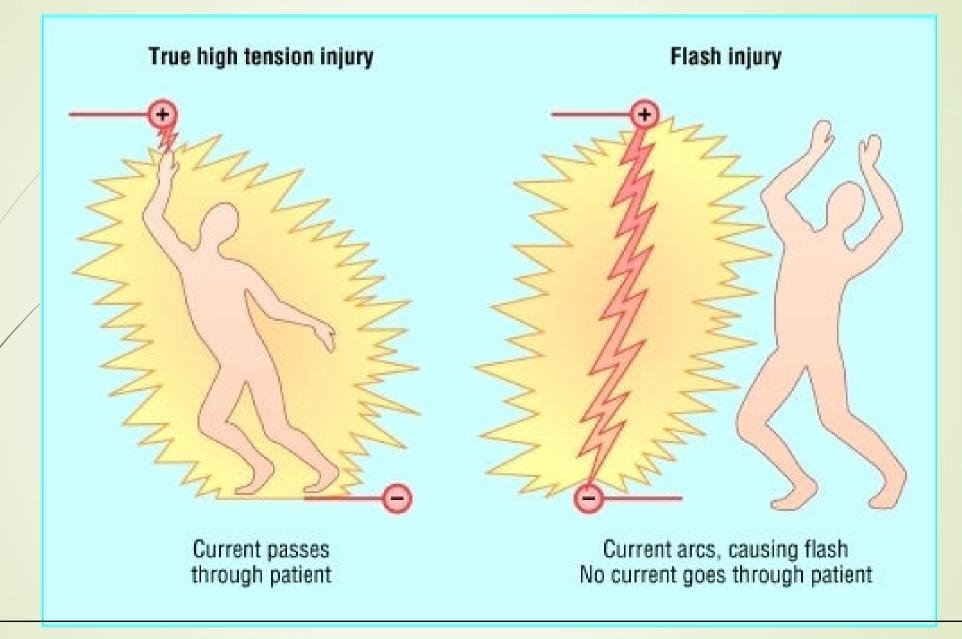






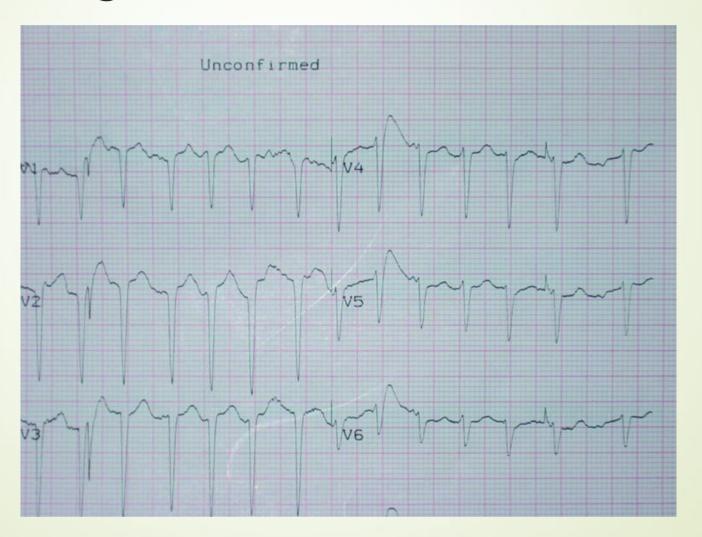
Electrical Burns

- caused by an electrical current passing from an electrical outlet, cord or appliance through the bodyfrom one point to another, creating "entry" and "exit" points.
- The tissue between these two points can be damaged by the current.
- Can be high voltage or low voltage injuries
- High voltage injuries:
 - Flash burns
 - True high tension injury





Electrocardiogram after electrocution showing atrial fibrillation



Chemical Burns

- caused by exposure to reactive chemical substances such as strong acids or alkalis
- These burns tend to be deep, as the corrosive agent continues to cause coagulative necrosis until completely removed.
- Alkalis tend to penetrate deeper and cause worse burns than acids.
- Cement is a common cause of alkali burns.





By the degree and depth of a burn

- First degree
- Second Degree
- Third degree
- Fourth degree

First degree Burn

- Cause: Flash flame. ultraviolet(sunbum)
- Surface appearance: Dry, no blisters, no or minimal edema
- Colour: Erythematous
- Pain level: Painful





Second Degree Burn (Superficial partial thickness)

- Cause: Contact with hot liquids or solids, flash flame to clothing, direct flame, chemical. Ultraviolet
- Surface appearance: Moist blebs, blisters
- Colour: Mottled white to pink, cherry red
- Pain level: Very Painful





Third degree Burns (Deep Partial thickness)

- Cause: Contact with hot liquids or solids, flame. chemical, electrical
- Surface appearance:Dry with leathery eschar until debridement; charred vessels visible under eschar
- Colour:Mixed white, waxy. pearly; dark, khaki, mahogany; charred
- Pain level: Little or no pain; hair pulls out easily





Fourth degree Burn

- Cause: Prolonged contact with flame, electrical
- Surface appearance:Same as third degree, possibly with exposed bone, muscle, or tendon
- Colour: Same as third degree
- Pain level: Same as third degree



- Currently burn depth assessment is done clinically
- Future: multisensor heatable laser
 Doppler flowmeter

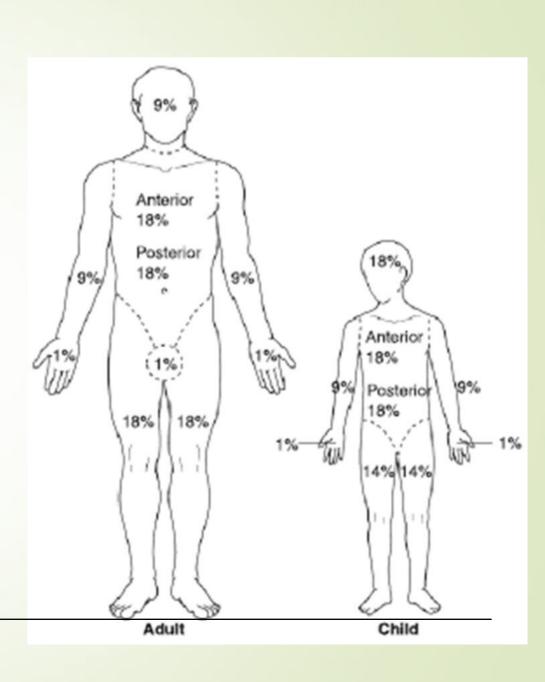




Assessing the extent of burn

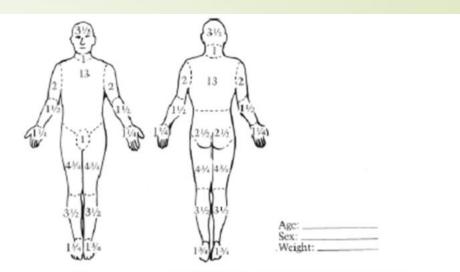
- This is calculated as a percentage of Total body surface area (TBSA)
- Rule of 9
- Lund and Browder chart

- The Rule of 9's provides a simple method of estimating
- total body surface area burned.
 Due to differences in body proportions,
- the percentage for each body area is different in adults and
- children.





The Lund and Browder chart provideo a more precise estimate of bum TBSA for each body part based on the individual's age.



Area	Birth-1 y	1-4 y	5-9 y	10–14 y	15 y	Adult	Partial thickness 2°	Full thickness 3°	Total
Head	19	1.7	13	11	9	7			
Neck	2	2	2	2	2	2			
Anterior trunk	13	13	13	13	13	13			
Posterior trunk	13	1.3	13	13	13	13			
Right buttock	21/2	21/4	21/2	21/2	21/1	21/2			
Left buttock	21/2	2 1/s	21/2	21/2	21/4	21/4			
Genitalia	1	1	1	1	1	1			
Right upper arm	4	4	4	4	4	4			
Left upper arm	4	4	4	4	4	4			
Right lower arm	3	3	3	3	3	3			
Left lower arm	3	3	3	3	3	3			
Right hand	21/2	21/1	21/2	21/2	21/2	21/2			
Left hand	21/2	21/1	21/2	21/2	21/2	21/2			
Right thigh	51/2	61/4	8	81/2	9	91/2			
Left thigh	514	61/4	8	81/2	9	914			
Right leg	5	5	51/2	6	61/2	7			
Left leg	5	5	51/2	6	61/2	8			
Right foot	31/2	31/2	31/2	31/2	31/2	31/2			
Left foot	316	31/1	31/2	31/2	31/1	31/2			
						Total			

Pathophysiology of Burn



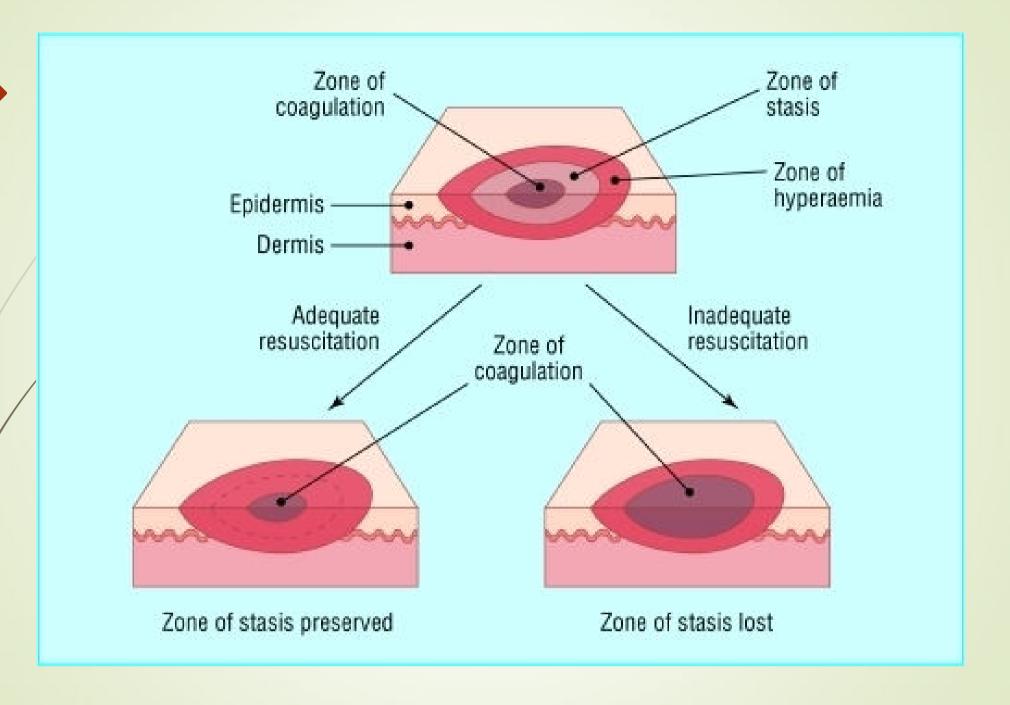
Body's response to burn injury

Burn injuries result in both local and systemic responses.

Local response

- The three zones of a burn were described by Jackson in 1947.
- Zone of coagulation—This occurs at the point of maximum damage. In this zone there is irreversible tissue loss due to coagulation of the constituent proteins.
- Zone of stasis—The surrounding zone of stasis is characterised by decreased tissue perfusion. The tissue in this zone is potentially salvageable. The main aim of burns resuscitation is to increase tissue perfusion here and prevent any damage becoming irreversible. Additional insults—such as prolonged hypotension, infection, or oedema—can convert this zone into an area of complete tissue loss.
- **Zone of hyperaemia**—In this outermost zone tissue perfusion is increased. The tissue here will invariably recover unless there is severe sepsis or prolonged hypoperfusion.
- These three zones of a burn are three dimensional, and loss of tissue in the zone of stasis will lead to the wound deepening as well as widening.





Clinical image of burn zones. There is central necrosis, surrounded by the zones of stasis and of hyperaemia





Systemic response

■ The release of cytokines and other inflammatory mediators at the site of injury has a systemic effect once the burn reaches 30% of total body surface area.

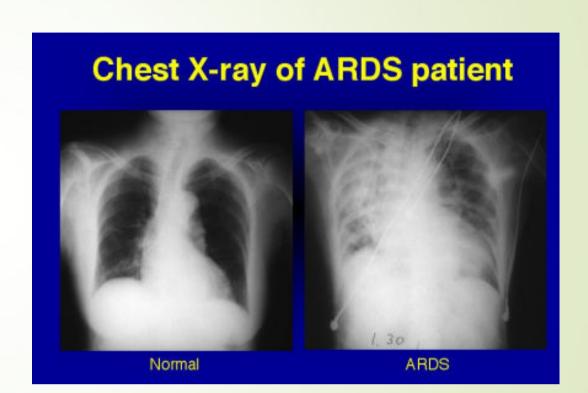
Cardiovascular changes

- Capillary permeability is increased, leading to loss of intravascular proteins and fluids into the interstitial compartment.
- Peripheral and splanchnic vasoconstriction occurs.
- Myocardial contractility is decreased, possibly due to release of tumour necrosis factor a.
- These changes, coupled with fluid loss from the burn wound, result in systemic hypotension and end organ hypoperfusion.



Respiratory changes

Inflammatory mediators cause bronchoconstriction, and in severe burns adult respiratory distress syndrome can occur.



Gastrointestinal changes

- Atrophy of the small bowel mucosa occurs within 12 hours of injury in proportion to the burn size
- reduced uptake of glucose and amino acids, decreased absorption of fatty acids, and a reduction in brush border lipase activity
- Intestinal permeability to macromolecules, which are normally repelled by an intact mucosal barrier, increases after a burn.
- splanchnic hypoperfusion occurs
- early and aggressive enteral feeding to decrease catabolism and maintain gut integrity.



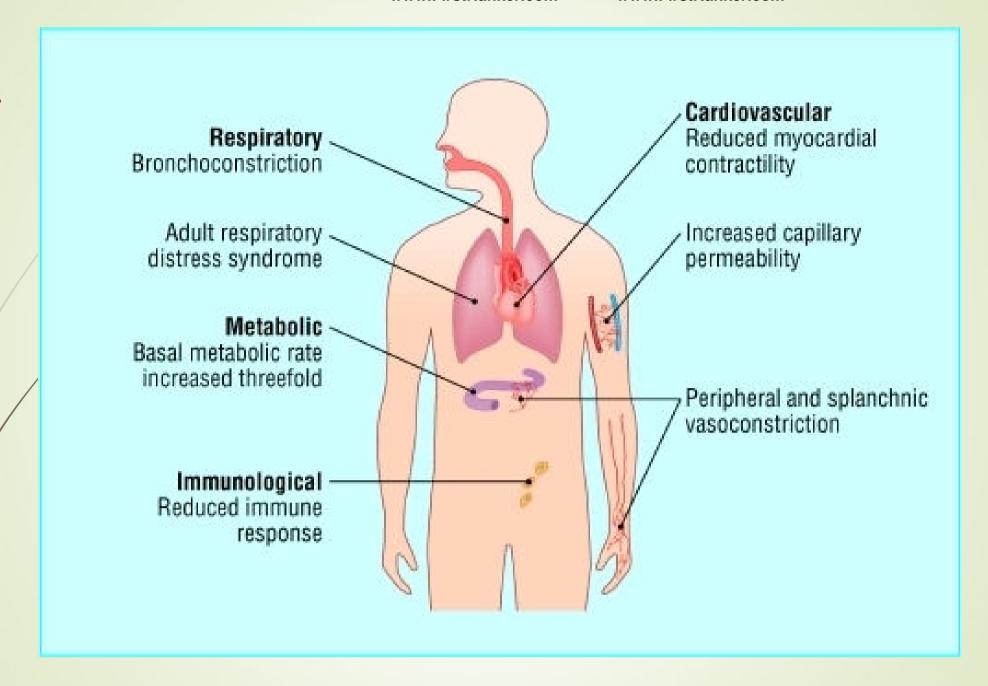
Metabolic changes

- The basal metabolic rate increases up to three times its original rate.
- A stress such as a severe burn induces the release of inflammatory hormones, which results in
 - gluconeogenesis,
 - lipolysis, and
 - proteolysis.

Inflammatory changes

Non-specific down regulation of the immune response occurs, affecting both cell mediated and humoral pathways.





Prevention



BURNS ARE PREVENTABLE

Prevention strategies: forming a burn prevention plan

- Prevention strategies should address the hazards for specific burn injuries, education for vulnerable populations and training of communities in first aid.
- An effective burn prevention plan should be multisectoral and include broad efforts to:
 - improve awareness
 - develop and enforce effective policy
 - describe burden and identify risk factors
 - set research priorities with promotion of promising interventions
 - provide burn prevention programmes
 - strengthen burn care
 - strengthen capacities to carry out all of the above.



Recommendations for individuals, communities and public health officials

- 1. Enclose fires and limit the height of open flames in domestic environments.
- Promote safer cookstoves and less hazardous fuels, and educate regarding loose clothing.
- Apply safety regulations to housing designs and materials, and encourage home inspections.
- 4. Improve the design of cookstoves, particularly with regard to stability and prevention of access by children.
- 5. Lower the temperature in hot water taps.
- 6. Promote fire safety education and the use of smoke detectors, fire sprinklers, and fire-escape systems in homes.

- 7. Promote the introduction of and compliance with industrial safety regulations, and the use of fire-retardant fabrics for children's sleepwear.
- 8. Avoid smoking in bed and encourage the use of child-resistant lighters.
- 9. Promote legislation mandating the production of fire-safe cigarettes.
- 10. Improve treatment of epilepsy, particularly in developing countries.
- 11. Encourage further development of burn-care systems, including the training of health-care providers in the appropriate triage and management of people with burns.
- 7. Support the development and distribution of fire-retardant aprons to be used while cooking around an open flame or kerosene stove.



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