13

Burns, Skin Grafting and Flaps

- Burns
- Skin grafting
- Flaps
- Electrical burns
- Chemical burns
- What is new? / Recent advances

BURNS

Definition

Burns is a type of coagulative necrosis caused by heat, transferred from the source to the body.

Frost bite which occurs in cold countries is also a coagulative necrosis but it is caused by extreme degrees of cold. Scald is a burn caused by moist heat (steam). Burns never occur at temperatures less than 44°C.

Types

- Thermal: Flame burns and scald burns
- Electrical
- Chemical
- Radiation

Pathophysiology of burns shock

Although the exact mechanism of the postburn microvascular changes and hypovolaemia leading to low cardiac output and poor tissue perfusion has not been determined, the following mechanisms have been proposed:

1. **Increased capillary permeability** leading to fluid and protein leakage from the intravascular space.
2. **Decreased plasma oncotic pressure** due to hypoproteinaemia resulting from loss of protein from the intravascular space.
3. **Increased capillary hydrostatic pressure** due to vasoconstriction or partial blockage of vessels w aggregate of cells and platelets.
4. **Reduced clearance of fluid and protein** from interstitital space by lymphatic ducts due to blockage platelet aggregates and fibrin clots.
5. **Intracellular fluid accumulation** due to impaired membrane function.
6. **Increase in osmotic pressure in the burned tis** leading to further fluid accumulation.
7. **Increased evaporative water loss**
8. **Depressed myocardial function**
   - Chemical mediators released from the site of injury responsible for the development of typical inflamma response. This results in rapid and dramatic oed formation.
   - The activated complement cascade system facili liberation of various permeability factors suc

KEY BOX 13.1

BURNS PATIENT AND THE KIDNEY

- Maximum water reabsorption by release of ADH f posterior pituitary as a compensatory mechanism stimulated.
- Aldosterone is released to cause maximum sod reabsorption.
- Acute tubular necrosis (ATN) can occur due to toxins.
- Injury to kidney can occur due to myoglobin.

Table 13.1

	Involved area	Pain	Adnexa	Appearance	Healing
I° Burn	Only epidermis	+	+	Erythema/oedema	3–5 days without scar
II° Burn	Varying depth of dermis	++/–	+	Blister, soft waxy, white, elastic, pain sensation to needle prick present.	10–20 days, with hypertrophic scar.
III° Burn	Involvement of entire depth of epidermis and dermis.	– –	Lost	Tough, dry, eschar, thrombosed subcutaneous vein. Pain sensation to needle prick absent.	3–5 weeks, eschar separates. **Always needs SSG.**

histamine, prostaglandins (PGF-1, PGF-2, PGF-2α) and thromboxane.

- Macromolecular leakage into burned areas, catabolism and reduced immunoglobulin synthesis results in a decreased concentration of all individual immunoglobulin levels and triggering of complement cascade.
- **Inhalation injury:** Glottic oedema, necrotising bronchitis, pneumonia are the dangerous events that follow an inhalation injury.
- **Septic shock:** Due to infection by micro-organisms. Infection commonly occurs from cross infection. GI system, genitourinary system, respiratory system, IV cannula site are various other portals of entry of infection.
- **Endotoxins:** Lipopolysaccharides and endogenous chemical mediators (cytokines) such as TNF-α and interleukins are activated resulting in systemic inflammatory response syndrome and multiorgan failure.

Metabolic response to burn injury

- **Gluconeogenesis** and glycogenolysis is increased.
- **Lipolysis:** It is increased and fatty acids are released. They are re-esterified into triglycerides resulting in fat accumulation in the liver.
- **Proteolysis:** The result is increased production of urea which is excreted in the urine. As a result of this, there is increased effux of amino acids from skeletal muscle pool including alanine and glutamine.
  Significance: Burns patient require more than 1g/kg/day proteins.
- Glutamine can also be given
- **Catecholamines** are massively elevated in burn injury.

Emergency care

- Any patient exposed to smoky fire should receive 100% oxygen *via* a non-rebreathing mask. If he is unconscious, endotracheal intubation should be performed.
- Intravenous line and administration of Lactated Ringer solution at 1L/h in adult.
- Transport the patient-warm and wrapped in a clean sheet. Clothing and jewellery should be removed because the swelling begins immediately.

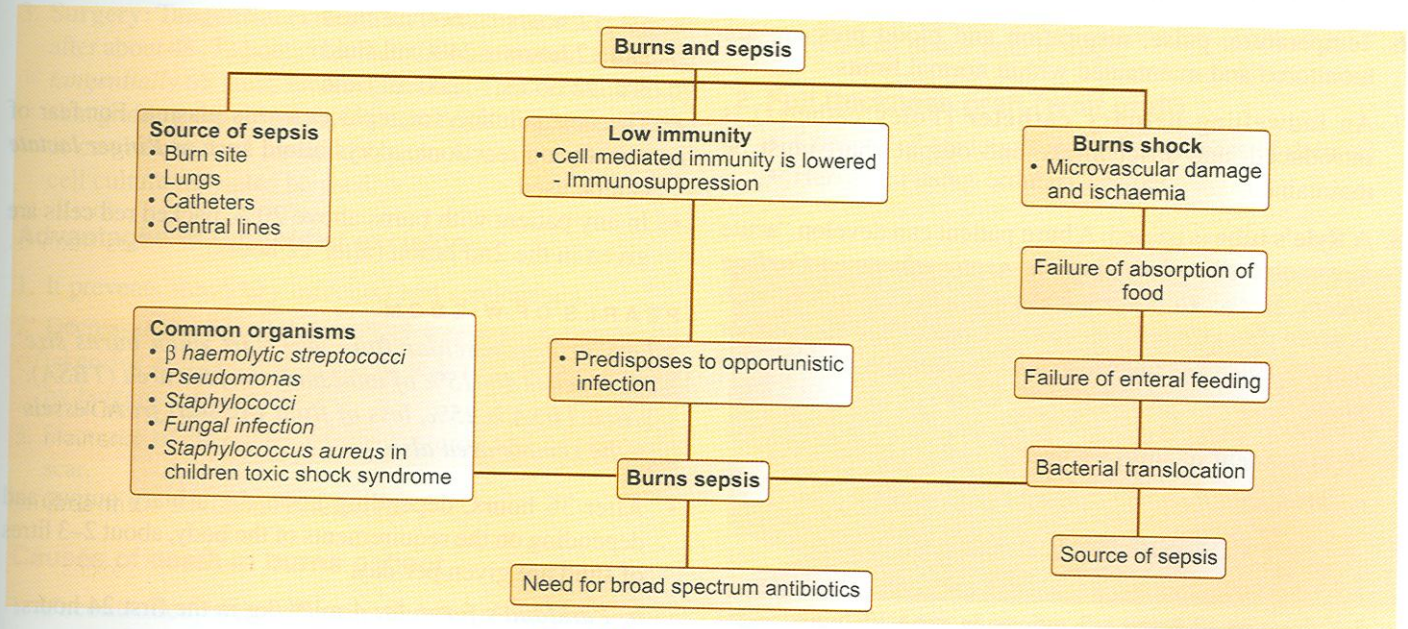


Fig. 13.1: Burns and sepsis

- Small burns can be managed by cool water. **Do not use ice-cold water.**

Management of burns patients

1. **First aid:** Cold water bath should be given immediately. This takes away the heat, stabilises mast cell, thus decreasing the release of histamine and reduces oedema.
2. Careful history-taking. **The time since sustaining the injury,** the type of heat source, location and circumstances of the burn should be recorded.
3. Hospitalisation and admission in a burns ward with air-conditioning facility.
4. **Assessment of depth of burns:** (Table 13.1)
   - **Partial thickness burns:** Here, superficial layers of skin are destroyed. Epidermis and variable portion of dermis are involved. Since the nerve endings are exposed, it causes severe degree of pain.
   - **Full thickness burns:** Involvement of full thickness of dermis with epidermis. Since the nerves are destroyed, it is less painful.
5. **Assessment of extent of burns in terms of body surface area (BSA):** It is calculated by a ***Rule of 9 "Rule of Wallace"***
   - Burns of head and neck: 9%
   - Burns of upper limbs: 9 × 2 = 18%
   - Burns of anterior trunk = 18%
   - Burns of posterior trunk = 18%
   - Burns of the lower limbs: 18 × 2 = 36% (front and back of each lower limb is 9%)
   - Burns of external genitalia: 1%
6. Temperature, pulse, respiration and blood pressure are monitored and maintained within normal limits.
7. An indwelling **urinary catheter** (Foley catheter) is introduced, and strict intake and output chart must be maintained.
8. A **Ryle's tube** is passed. A burn patient can develop "acute stress ulcers" called as ***acute peptic ulcers*** or ***Curling ulcers***. Hence, to prevent bleeding, a cold stomach wash is given through the Ryle's tube. Antacids and H_2 receptor blockers such as Ranitidine 150 mg twice a day are also given.
9. **Replacement of fluid volume**

   a. Muir and Barclay formula

   $$1 \text{ ration} = \frac{\% \text{ of burns} \times \text{body weight (kg)}}{2}$$

   3 rations in 12 hours, 2 rations in next 12 hours and 1 ration in next 12 hours.

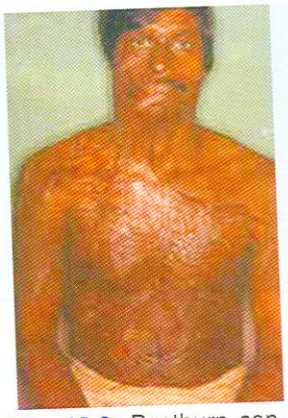


Fig. 13.2: Postburn contracture in an adult

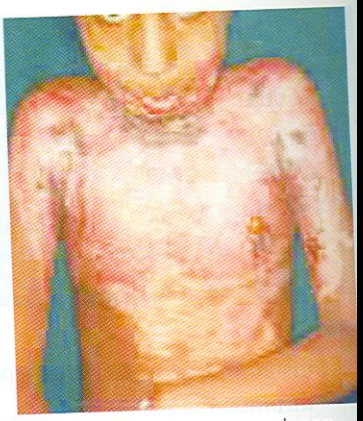


Fig. 13.3: Postburn neck contracture in a child

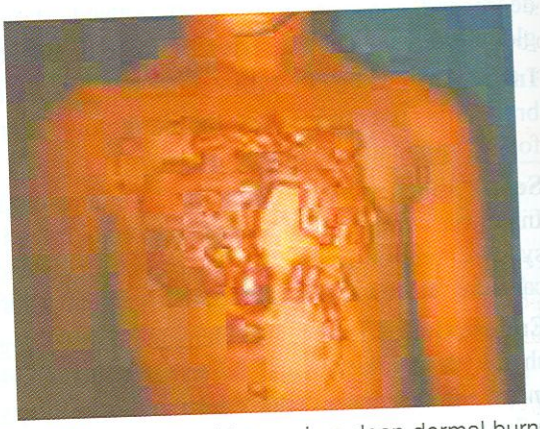


Fig. 13.4: Hypertrophic scar in a deep dermal burns

Example: 40% burns patient weighing 60 kg.

$$1 \text{ ration} = \frac{40 \times 60}{2} = 1200 \text{ ml}$$

1st 12 hours : 3600 ml fluid
2nd 12 hours : 2400 ml fluid
3rd 12 hours : 1200 ml fluid

- The best solution for replacement is plasma. For fea transfusion reactions, a crystalloid such as ***Ringer lac*** can be used.
- In any patient with burns above 20%, packed red cell given in the 2nd ration (after 12 hours).

PEARLS OF WISDOM

Loss of intravascular fluid will start when burns s approaches 10–15% of total body surface area (TBS When TBSA is 25%, loss of fluid will start from ves in the remote area also.

- After 36 hours, depending upon the urinary outpu depending on the requirements of the body, about 2–3 of fluid are given per day.

b. Parkland's formula: 4 ml/%/kg in the first 24 ho 1/2 : In 1st 8 hours, 1/4 : Next 8 hours each.

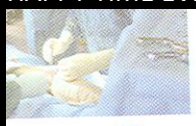


Fig. 13.6: Skin graft is taken from thigh using Humby's knife

Fig. 13.7: Skin graft is applied over the recipient area

- Needs excellent vascularity of the recipient wound for graft survival.
- Used for small uncontaminated wounds produced after excision of skin lesions or after release of skin contractures (lower eyelids, fingers).
- **Donor area needs primary suturing or split skin graft** for healing and hence limits the size of the graft.
- Unlike split skin grafts, full thickness grafts do not contract and retain their colour. Hence, they are cosmetically superior.

Indications of skin graft (Key Box 13.3)

1. Skin loss

- **Post-traumatic** (e.g. avulsion and degloving injury)
- **Post-surgical** (e.g. excision of tumours, excision of burn wound)
- As a result of **pathological process** (e.g. venous ulcer, diabetic ulcer)

2. Mucosa loss

- After **excision** of lesions of oral cavity, tongue.
- For **resurfacing** reconstructed vagina in cases of vaginal agenesis.

Contraindications for skin graft

- Infection by ***beta-haemolytic streptococci***. They produce fibrinolysin which dissolves fibrin.
- Presence of an **infected wound** with copious discharge in the vicinity.

KEY BOX 13.3

IDEAL REQUIREMENTS FOR FREE SKIN GRAFT

1. Wound should be free from infections such as *Streptococci* and *Pseudomonas*
2. Vascular wounds, e.g. wounds with healthy granulation.
3. Wound should be thoroughly debrided.
4. Haemostasis must be achieved before placing the graft.
5. Close and immobile contact between graft and the wound.
6. Recipient area should be immobilised with **POP** slab.

- **Avascular wounds:** with exposed bare bone without periosteum, exposed tendon without **paratenon** and exposed cartilage without perichondrium.

Healing of the donor area

- Donor area of split skin graft heals by epithelialisation from the adnexal remnants of dermis, pilosebaceous follicle and/or sweat gland apparatus. Complete healing of donor area occurs by 8–10 days.

The process of graft 'take'

- The processes which result in **reattachment** and **revascularization** of the graft to the bed are collectively referred to as **"take" of graft**.
- The graft initially adheres to its new bed by fibrin. Revascularisation starts by 48 hours and is completed by 4–5 days. This is achieved by the outgrowth of capillary buds from the recipient area to unite with those on the deep surface of graft. For the first 2 days after grafting, the skin graft derives its nutrition from the wound by the process of serum imbibition/plasmatic circulation.

FLAPS

Flap is a block of tissue transferred from donor to recipient area along with its vascularity.

Common indications for flap surgery

- To cover defects/wounds **where free skin graft cannot be used, e.g. exposed bare bones, bare tendons, bare cartilage.**
- Wounds with exposed joints, exposed major vessels and nerves.
- **Implant exposure** following orthopaedic procedures.
- In wounds with **soft tissue loss**, where future reconstructive surgery is contemplated.
- Defects which need better contour to improve cosmesis.
- Breast reconstruction following mastectomy.

Classification of flaps

These are broadly classified into Pedicled flaps and Free flaps.

I. Pedicled flaps

Pedicle or the base remains attached to the donor site during its transfer to the recipient area. Pedicled flaps may be of following types:

1. **Local flaps**, e.g. rotation, transposition, limberg and bilobed
2. **Regional flap**, e.g. PMMC, DP for head and neck defects, TRAM for breast reconstruction.
3. **Distant flaps**, e.g. groin flap, subaxillary flap for hand defects.

A few examples of the pedicled flap

- Skin flap
- Fasciocutaneous flap
- Muscle flap
- Myocutaneous flap
- Adipofascial flap
- Osteocutaneous flap

II. Free flaps

These are completely detached from the donor area before being transferred to the recipient area. The vascularity of the flap at the recipient site is immediately restored by anastomosing the vessels of the flap with the vessels at the recipient area using microvascular techniques.

Some of the commonly performed flaps

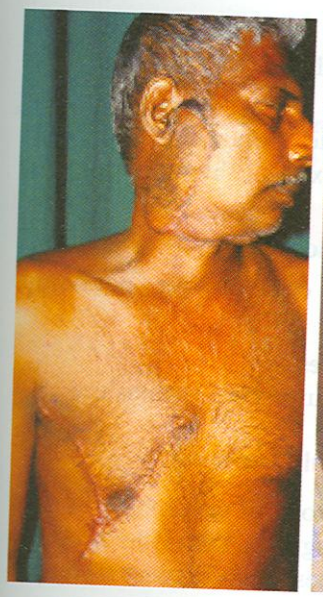


Fig. 13.8: PMMC flap following radical parotidectomy

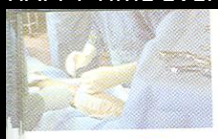
Fig. 13.9: Latissimus dorsi flap used to cover the defect in the arm following radical excision of a sarcoma

Fig. 13.10: Bedsore covered with local advancement flap

- **Forehead flap:** Entire forehead skin can be raised based on anterior branch of superficial temporal artery. It bears an unsightly scar of the donor site. **Median (Indian) forehead flap** based on supratrochlear vessels is a very useful flap in reconstructing defects over nose.
- **Deltopectoral flap (DP):** It is supplied by upper 4 perforating branches of internal mammary artery and is used to reconstruct defects of neck and lower face. After about 4 weeks, the flap is divided and the base is returned to the chest wall.
- **Pectoralis major myocutaneous flap (PMMC):** Pectoral branch of thoracoacromial artery is the pedicle of this flap. It is the ideal pedicled flap for reconstruction of head and neck defects following ablative surgeries for various head and neck cancers. Hence, it is described as a workhorse among the flaps. Osteomyocutaneous PMMC **flap by including 5th or 6th rib can be used for mandibular reconstruction.**
- **Latissimus dorsi flap (LDF):** As a myocutaneous flap, based on thoracodorsal vessels can be used for reconstruction of the lower half of face, neck, breast, chest wall, axilla, and upper arm.

Free (Microvascular) flaps

Using operating microscope and microvascular techniques, it is possible to do a free tissue transfer of tissues such as skin, muscle, bone, intestine, omentum, etc. The procedure involves anastomosis of vessels of the flap to vessels at the donor site, e.g. latissimus dorsi muscle myocutaneous flap, radical artery forearm flap, gracilis flap, free fibula flap, etc.



ELECTRICAL BURNS

The most important factors taken into consideration while assessing the damage caused by the passage of electrical current through the body are: **the nature of electric current and the resistance of body tissues through which the electric current is passing.**

- When a portion of the body comes in contact with a live electric wire, the actual point where the electric current enters the body is known as point of entry and where it is grounded as the point of exit. The most important factor which decides the severity of injury is the **"voltage"** of current.
- The resistance offered by the tissues to the flow of current leads to the **conversion of electrical energy to thermal injury** and causes tissue damage.

Types of injuries

- **True electrical injury:** The burns is as a result of heat generated due to the passage of current.
- **Flame burns:** It is due to an electrical flash or spark.
- **Arc burns:** Localised injury due to intense heat at the termination of current flow. It occurs when it jumps the gap between the source and the conductor, e.g. flexor aspect of joints.

Classification

A. Acute injuries

I. Burn injuries due to electric current itself

a. Low voltage injuries (less than 1000 volts of current)
(i) Flash burns (ii) Contact burn.

b. High voltage injuries (more than 1000 volts of current)
(i) Flash (ii) Arc or contact
1. Punctate
2. Extensive
3. Extensive with vascular impairment of extremities.
   - Compartment syndrome, Dry gangrene

II. Other associated injuries

a. Thermal burns due to ignited cloth
b. Acute CNS problems
c. Acute peripheral neuropathy
d. Cardiac/respiratory arrest
e. Injuries to other internal organs due to fall or electric current.

B. Delayed injuries

1. Delayed spinal cord injury
2. Delayed peripheral neuropathy
3. Optic nerve atrophy, cataract

Summary (Key Box 13.4)

KEY BOX 13.4

SUMMARY OF MANAGEMENT OF ELECTRICAL INJURY

- Initial resuscitation as in thermal burns
- Low voltage injury
  Superficial—conservative
  Deep dermal—tangential excision and split skin graft
  Contact burns—excision with SSG/flap
- **High voltage injury**
  Flash burns: Superficial—conservative
  Deep dermal: Tangential excision and split skin graft
- **Arc or contact burns:** Vascular compromise—immediate fasciotomy, amputation
- **Punctate wounds:** Excision and grafting
- **Extensive injury without vascular compromise:** Wider excision with split skin graft or flap cover
- Recipient area should be immobilised with **POP** slab

CHEMICAL BURNS

- Chemicals are a relatively uncommon cause of burns. The chemicals used in industry, science laboratories and at home are the usual agents.
- The tissue damage in chemical burns is mainly due to prolonged contact period and effects of systemic absorption.

Classification of agents that cause injury

1. **Acids** : Hydrochloric acid, sulphuric acid, nitric acid hydrofluoric acid, phenol (carbolic acid), oxalic acid
2. **Alkalis** : Sodium hydroxide, potassium hydroxide ammonium hydroxide, lithium, barium and calcium hydroxide
3. **Others** : **Inorganic substances**
   Phosphorus, wet cement lime, potassium permanganate
   Organic substances
   Kerosene, petrol, turpentine, naphthalene.

Modes of action of chemicals on tissues

- **Acid** causes coagulative necrosis of the skin due to rapid conversion of protein to coagulum salt of the acid. The coagulated eschar prevents these acids to penetrate deeply. Activity in the tissues continues for a long time.
- **Alkalis** are corrosive agents and produce extensive denaturation of tissue proteins. These produce more tissue destruction than acids.

First aid

- **Early irrigation** of injured area with large volume of water or running water (with few exceptions) is the main focus in the first aid management of chemical injuries. Hydrotherapy mechanically cleans the area reducing the concentration of chemicals and duration of contact. Earlier the hydrotherapy is started, more is the benefit obtained.
- Since the absorption of phenol increases with dilution, surface phenol is removed by solvent **polyethylene glycol before hydrotherapy** is started.

Definitive wound closure

Achieved in a similar way as in any other thermal injury

- **Primary excision and split skin grafting,** of all acid and alkali burns, if done before 10 days.
- **Grafting of granulating wound**, if the patient comes late, with wound infection. This takes much longer (1½ months) compared to thermal burns (3 weeks).

OCULAR INJURIES

Very common in acid and alkali burns. Severe blepharospasm and forceful rubbing increases the severity of injury. Various sequences of events are

- Sloughing of corneal epithelium
- Stromal oedema
- Corneal ulceration
- Perforation
- Panophthalmitis

- If perforation does not occur, corneal opacity and heavy vascularisation occurs. Quick, thorough and prolonged lavage of cornea with water is the most important measure in the first aid treatment. Systemic and topical steroids minimises inflammation and scarring of cornea.

Miscellaneous

SKIN SUBSTITUTES

- This is one of the important requirements wherein large surface area of burns and skin is lost.
- An ideal substitute must be affordable, permanent, provide normal pigmentation, resist scar formation and grow with developing children.
- Few examples of skin substitutes are given below:
  - **Dermal substitutes:** They allow for creation of a 'neodermis'. They are formed from patient's own mesenchymal cells. Once 'neo-dermis' is formed, split thickness skin graft is applied. Thus, the burns site is closed quickly with less scarring.
  - **Cultured epithelial autograft** is another example of dermal substitute. They are cultured from patient's own full thickness biopsy. It will require 3 weeks to grow.
  - Another cultured skin is a **biologic dressing** from cultured **neonatal keratinocytes** and **fibroblasts.** They are all very expensive.

WHAT IS NEW IN THIS CHAPTER? / RECENT ADVANCES

- Burns and sepsis have been discussed in more detail.
- Skin substitutes have been added.

* All photographs and the entire text in this chapter are contributed by Professor Pramod Kumar, Head of the Dept. of Plastic Surgery, KMC, Manipal and Dr Bhaskar KG, Senior Consultant, Dept. of Plastic Surgery, Medical Trust Hospital,Cochin, Kerala.