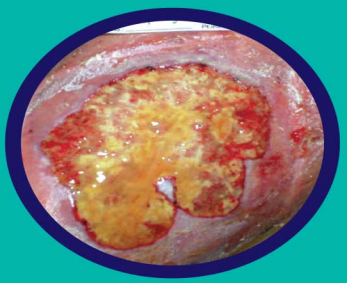
Raj Mani
Luc Téot



The Basic Needs to Achieve WOUND HEALING







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Management of Burns: Step-by-Step Approach to the Burn Wound

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INTRODUCTION

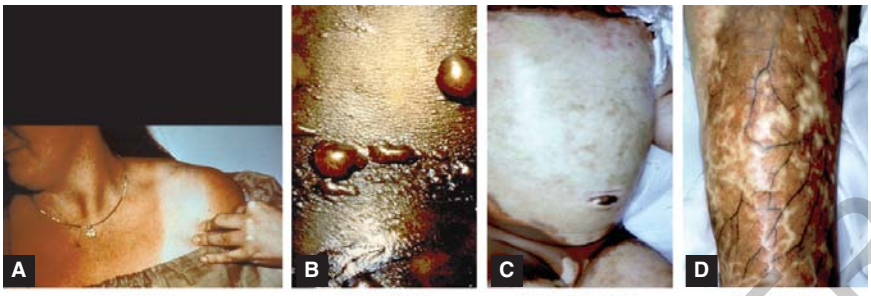
Burn injury is one of the most devastating type of trauma that is encountered today. Burn injury can occur due to flame-fire, hot liquids, electricity, corrosive chemicals and radiation. Though a preventable condition, incidence is increasing due to modern warfare and terrorist attacks. Patients go through a phase of initial shock, accompanied by pathophysiological changes in the system, and various types of burn wound manifestations, that without timely intervention can be fatal in some cases. The extent and depth of the burn wound are both significant factors that contribute to the ultimate mortality and morbidity of the victims.

Today, critical care medicine has opened out avenues of managing the problem of shock, ARDS, pulmonary complications, respiratory failure and renal failure. But, inspite of such adequate burn management, infection of burn wound is still a problem to treat and the evolution of the burn wound literally predicts the outcome.

EVOLUTION OF BURN WOUND

Human skin can tolerate temperatures upto 40° C (104° F) for relatively long periods of time before apparent injury. Temperature above that level, however, produces, logarithmic increase in tissue destruction. The depth of burning is determined by a combination of the burning agent, the temperature and the time of exposure.

Burns are classified as first, second and third degrees with respect to depth, and more descriptive terms being partial thickness and full thickness burns. Despite modern technology, clinical observation still remains the gold standard.



Figures 3.1A to D: (A) Epidermal burns over the neck due to Sunburn—shows only erythema. (B) Superficial partial thickness burn over the abdomen—showing typical blisters. (C) Deep partial thckness burn over the abdomen—white parchment like area showing deeper element of burns. (D) Deep III degree burn, where full thickness of the skin is burnt and the clotted blood in the veins are seen

CLASSIFICATION OF DEPTH OF BURNS (FIGS 3.1A TO D)

FIRST DEGREE

Involves epidermis only, has mild erythema like sun burn and peals off in 2 weeks.

PARTIAL THICKNESS (SECOND DEGREE)

Involves entire epidermis and part of the dermis that is involved in dermal necrosis. Blistering and oedema are seen immediately.

DEEP BURN (THIRD DEGREE)

White to black hard "leathery" inelastic eschar is seen which is anaesthetic, and thrombosed vessels are seen in the depth.

Depending on the depth and extent, the pathophysiological and biochemical changes takes place in the system.

PATHOPHYSIOLOGICAL AND BIOCHEMICAL CHANGES IN BURNS

The protective covering of the skin helps maintain fluid and electrolyte balance by preventing the loss of fluid to the environment. Water loss occurs in the sweat and through the respiratory tract.

Burn injury occurs as a result of an energy transfer from heat source to the body. Individuals exposed to heat energy source sustain thermal injury characterized by cell death and coagulation necrosis of the tissues. The temperature and duration of the exposure dictates the injury to

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the tissues. The initial cessation of blood flow in both arterial and venous systems recovers within 24 to 48 hours in partial thickness burns but persists for 3 to 4 weeks in deep burns, and by that time dead tissue necroses, and gets infected.

There is also marked increase in the capillary permeability, which is maximum within 24 to 30 hours, and major segment being within 12 hours, which results in oedema. As the oedema settles within 48 hours in a well-hydrated patient, macrophages appear in the scene. Invading colonised bacteria secrete proteases and collagenases, which separates live tissue from the dead tissue. Superficial burn epithelialises within 10 days and superficial partial thickness burns also heals well within 2 weeks if covered adequately by biological dressings. Deep partial and deep burns needs early intervention by way of excision and grafting.

EVOLUTION OF THE BURN WOUND

Acute phase	:	Post-burn oedema and blister formation in the inflammatory phase.
Proliferative phase	:	Fibroplasia.
Restoration phase	:	Epidermis and dermis forms.
Maturation phase	:	Wound contracture and scar contracture occurs.

INFLAMMATORY PHASE

All wounds respond to injury with an inflammatory phase. The stage is characterised by initial homeostasis followed by the inflammatory process with migration of PML: Polymorph—Nuclear Leucocytes, macrophages and lymphocytes. This phase has the teleological purpose of protecting the tissue from further injury and from microbial invasion. The inflammatory phase persists as long as the wound is open, and the intensity of inflammation also contributes to greater scarring.

Acute Burn Wound

Massive tissue oedema after burns is a complex, well recognised entity. This is seen in the burnt tissue and in the normal tissue. The latter may well be the result of the hypoproteinaemia caused by protein loss into the burn tissue. Primary process is a severe alteration in the integrity of the microcirculation. To this, an alteration in the cell membrane permeability to sodium ion occurs, which settles in the cell with water, producing swelling of the cells. Tissue and vascular ischaemia also produces tissue oedema. Many biochemical mediators are released from

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burnt tissue such as vasodilators and constrictors, prostaglandins, kinins, serotonin, histamine, oxygen free radicals, and various lipid peroxidases. These also play a part in burn oedema formation. The oedema usually settles after 48 hours, with systemic therapy for the shock, and by the use of biological dressings and topical application of heparin solution.

Oedema (Figs 3.21A to D)



Figures 3.2A to D: Child with facial burns and respiratory burns (A) Acute phase with massive oedema of the face. (B) 48 hours after burns. (C) Seven days postburn—Oedema is reduced and the biological membrane is dry. (D)The oedema has resolved and the granulation tissue is seen over the face

Blister Formation (Figs 3.3A and B)

Blisters occur due to entrapment of burn tissue fluid in partial thickness burns. Blister is not a closed wound as was once thought. The blister fluid has harmful inflammatory mediators.



Figures 3.3A and B: Two patients showing blister on the dorsum of the hand indicating superficial partial thickness burns

PROLIFERATIVE PHASE

If the acute burn wound is not excised and primarily closed, the wounds begin to granulate. Granulation tissue is very complex, consisting of pro-inflammatory cytokines, fibroblasts and capillary networks. It looks red, vascular and easily bleeds to touch. It has been postulated that

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low partial pressure of oxygen (PO_2), and reduced blood pressure stimulates the fibroblasts and macrophages to migrate into the wound site and produce irregular arrangement of collagen. Unrestricted growth of granulation tissue produces scarred contracted scars in a protracted phase.

RESTORATION PHASE

Restoration of epidermis and dermis occurs. The migration of the epidermal cells occurs from the unburnt skin appendages. In large wounds skin grafting accomplishes this.

Dermal healing is brought about by fibroblasts, which synthesise collagen, glycoproteins and mucopolysaccharides, that comprise the dermal fibres that give tensile strength to the wound. Today the origin of the fibroblast is known to be from Fibrocytes that are found in the adjacent loose subcutaneous tissue, adventitia of smaller blood vessels or in the cells in the fatty tissue.

MATURATION PHASE

This is the remodeling phase of healing. Collagen fibres organise themselves and line up parallel to the lines of stress. Process lasts for several months.

Wound Contraction and Scar

Wound contraction is defined as moving in from the margins of the wound to the centre. Then the cells migrate to the centre from the periphery to close the wound. This phenomenon is called "Picture trauma theory" of burn wound closure. Burn wound contraction occurs mainly due to the myofibroblasts that are present in the wound.

Burn wound contraction often continues with respect to tissue, beyond the actual closure of the wound. In such situations, sometime the scar becomes thick, contracts and there is functional and cosmetic disability to the patient. The contractile myofibroblast seems to be responsible for this phenomenon.

GROWTH FACTORS IN WOUND HEALING

Cells communicate with each other through specific molecules, which are usually proteins. Proteins that are used for cellular communication are called cytokines. These are secreted in minute quantity from one

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cell and then attach to a cellular receptor in another cell to cause a change, in the recipient cell. 'Growth factors' are cytokines that specially stimulate proliferating cells, and thereby help wound healing. In the burn wound, healing, acceleration of the donor sites, and partial thickness wound healing can be achieved. Those burn patients who benefit maximum are those wherein topical growth factors are applied over extensive burn wounds and over large donor sites. Shortening the time for the donor site reharvest by even by a few days would add up to significantly shortened hospital stay when multiple grafting procedures are required. In extensive burns one may end up with lack of large split thickness donor sites, and areas of chronic non-healing ulcers of burns. PDGF (Platelet Derived Growth Factor), EGF (Epidermal Growth Factor), FGF (Fibroblast Growth Factor) and TGF (Transforming Growth Factor) improve these chronic ulcers and delayed wound healing. Hence cytokines play an important role in burn wound healing.

MANAGEMENT OF BURN WOUND

The first step is to evaluate the burn wound in two surveys. The primary and secondary survey, primary being devoted to life-threatening conditions and secondary which is an elaborate survey, identifying the depth, surface area, presence or absence of shock and inhalation burn after identifying the general measures of resuscitation are done.

As far as the burn wound is concerned, attention is directed towards.

a. Preventing of the occurrence of further damage by cooling and washing with clean water.
b. In chemical burns, neutralisation of the chemical carefully by neutralising agents, or washing copiously with cold water is done.
c. Pain is controlled with morphine in extensive burns, and for cleanings the wound propofol (Braun, Germany) and ketamine hydrochloride are used for anaesthesia.
d. Ketamine hydrochloride is a dissociative anaesthetic agent most suitable for paediatric use. It is given in the dose of 1-1.5 mg/kg body weight intravenously as an analgesic.
e. Propofol is 2-6 di-isopropyl phenol, a short acting general anaesthetic agent with rapid onest of action and quick recovery.
f. Topical antimicrobial agents in an emollient base are used for simple small burns.
g. If there is a constricting eschar with distal oedema, liberal escharotomy and fasciotomy are done (Fig. 3.4D).

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Figure 3.4: III degree burns of the leg and foot due to high-tension electric current—Escharotomy done for releasing vascular compromise

h. Dressing the wound is a priority, but each burn centre adopts a policy best suited to its circumstances. The wounds are dressed with absorbent dressings, or antibiotic tulle dressings. In hot countries, exposure method of treatment and biological dressings are used extensively.

MANAGEMENT OF OEDEMA

Restoration of fluid balance, and reversal of the shock phase are ideal for the reduction of post-burn oedema. When there are obvious reductions of haemoglobin and plasma protein, these are replaced. Adequate urinary output also is an indicator of improvement, which reveals that increased intravenous (IV) pressure is reducing and the oedema fluid is getting reabsorbed.

MANAGEMENT OF BLISTERS

Blisters have to be deroofed for two reasons. One it could get punctured accidentally when the patient turns and second the blister fluid has lots of proinflammatory cytokines that are harmful and injurious to healing. This fluid has to be removed. After de-roofing, one will find the deep partial thickness burn, which can be dressed.

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DEEP PARTIAL THICKNESS AND DEEP BURNS (FIGS 3.5 TO 3.9)

These have to be treated in a different way. To make them less susceptible to bacterial invasion a thick film of topical antimicrobials are applied. The early removal of totally devitalised tissue and coverage with autografts is the ideal and gold standard today.

BURNS IN SPECIAL AREAS

Eye, hand and ear burn required immediate attention. An eye burn is treated by immediate tarsorrhaphy, or a soft lens is applied to the cornea to protect it.

BIOLOGICAL DRESSINGSM

In developing countries both acute burn and burn awaiting excision are protected by biological dressings. The ones commonly used are human and bovine amniotic membrane, native and reconstituted, bovine collagen membrane, plain and reconstituted from the tendo-Achilles of discarded cattle in leather industry as well as silver sulfadiazine incorporated collagen membrane. Alloderm™ and intergra are extensively used as skin replacements. However, these are costly, hence are not used in developing countries. The collagen membrane is an excellent biological dressing, and can be used for all superficial and superficial partial thickness burns. It can also be used as protection till excision is done.



Figure 3.5: Deep partial thickness burns, covered with reconstituted collagen membrane produced at the Central Leather Research Institute, Chennai: *A laboratory of Government of India*. These pictures show the way the collagen dressing adheres to the wound and healing takes place by 12th day of application. The final picture shows complete epithelialisation

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Figure 3.6: A child with superficial partial thickness burns due to bursting of cracker. Dressed with amniotic membrane. Healing is satisfactory after 8 days



Figure 3.7: Child with deep partial thickness flame burns dresssed with collagen membrane, after spraying the wound with topical low molecular weight heparin (dose of 400 IU/kg). Healing and epithelialisation are satisfactory. Heparin enhances wound healing

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Figure 3.8: Chronic burns—Deep partial thickness dressed with topical anti-microbial silver sulphadiazine cream. Epithelialisation occurs in 4 to 6 weeks



Figure 3.9: Deep partial thickness burns was excised tangentially and primary split skin graft was applied

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FULL THICKNESS BURNS

Small areas of full thickness burns due to electricity or chemicals are primarily excised and covered immediately by split skin graft or a flap.

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Larger areas of deep burns are excised in stages. Not more than 15 to 20% are excised each day. If autograft is available it is used to cover the wound, otherwise homografts or sterile collagen dressings are used. Once the general condition of the patient improves, autografting is done.

Primary reconstructions also are done using microsurgical free flaps. The scars are taken care off by massage with cortisone cream or with silicone gel and pressure therapy. Anaesthetic considerations are always thought about while planning the surgery. Ultimately, even if the burn is healed, the quality of the scar will dictate the psychological rehabilitation that the patients require.

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The Basic Needs to Achieve WOUND HEALING

Raj Mani and Luc Téot have both contributed to wound healing working individually and working as a team for several years.

Raj Mani has strived to bring measurements and, therefore, science into wound healing.

Luc Téot has energetically brought the understanding of technology to wound healers thereby improving wound management.

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