BURNS—THE CHALLENGE OF 1975

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The Burns régime at Baragwanath is not uniform and so there is an opportunity to evaluate various types of treatment.

CLASSIFICATION: This is based on the depth of skin burning, viz.

1. **Superficial** burns are grouped into two sections. The first being **first degree** burns which manifest as an erythema due to vaso-dilatation of the subepidermal vascular plexus
   The second being a vesicular or **second degree** burn which is manifested by the presence of large vesicles in consequence of cellular necrosis and the aggregation of interstitial fluids to produce blisters.
2. **Deep** burns are those where there is an epidermal destruction and this is subdivided into:
   a. **Deep dermal** burns indicating that epidermal-appendage remnants such as hair follicles, sweat and sebaceous glands persist and are therefore able to regenerate an epithelial covering albeit, often of poor quality.
   b. **Full thickness** burns. In these there are no epidermal remnants so that only peripheral regeneration is possible. The further implication is that fat is partially or completely destroyed and deeper structures such as tendons, muscles, neurovascular elements and bone may also be implicated in the injury.

The **causative factors** may be moist heat such as steam, dry heat such as flame and contact with hot metals, chemical and electrical agents. Each of these factors may hint at the depth of the burn but ultimately the defiinitive factor is the duration of contact with the burning agent.

PATHOLOGICALLY the sequelae of burns amounts to:

a. Cellular injury producing a reversible cloudy swelling.
b. Cellular necrosis manifesting its presence by the slough or eschar.
c. Impairment of cellular membrane function leading to the extravasation of serious fluid to the interstitial compartment, which in turn produces an oedema and this leads to a progressive circulatory fluid-volume depletion which in turn manifests itself in a state of circulatory shock.

For the purposes of resuscitation we involve the Rule of 9's to assess the body surface area involved.

On the basis of this assessment of the surface area which has been burnt we endeavour to restore the circulating blood volume using the formulae:

3 ml. x % area burnt (but never exceeding 30%) x mass in kgms. for the first 42 hours and administer thick or thin fluids depending on haematocrit evaluations.

In the first 6 hours a third of the volume is given as plasma or Ringer's lactate, thereafter the 2nd third of the volume is usually administered as plasma in the ensuing 12 hours, and the final third of the volume is given over 24 hours either as plasma or blood.

Over and above this intravenous replacement, normal daily oral fluid requirements are given and amount to 2 000-3 000 ml./24 hours.

Resuscitation progress is deemed adequate if a urinary output of 50-70 ml./hour is maintained.

Within 48 hours a diuresis usually follows and the oedema subsides fairly rapidly.

The commonly associated haemoglobinuria is due to haemolysis following the exposure of the red cells to heat. Haemoglobinuria may result in a physical blockage of the renal tubules and in turn this may lead to renal failure. For this reason it is essential to maintain early adequate renal perfusion in order to gain a "flushing" of the renal tubules.

In consequence of the enormity of the injury involving breakdown and repair, it is essential to endeavour to keep the patient in positive nitrogen balance and so a high protein, high calorie intake must be achieved. The need is underlined when one appreciates that an individual with 40% burns requires 7 000 cals./day and to provide adequate calories and proteins one would need to consume 38-40 eggs per day. The stoical Israelis are subjected to such a dietary intake.

Burns dressings are largely determined by the hospitalisation facilities and fall in three categories:

a. **Open method.** No dressings or medicaments are applied—however, clean air-conditioned cubicalisation is essential.
   The advantages are tremendous in as much as nursing care is minimal and early relatively free and full mobilisation is possible. However, with thick dry eschars flexural movement may be restricted.

b. **Semi-open method.** Here a layer of Tullegras is covered with an anti-bacterial agent such as:

Sulfamylon		Eusol	
Silver Sulphadiazine	Creams	Saline	Solutions
Gentamycin	or	Hibitane	
Aserbine		Silver nitrate	

   The presence of moisture permits a greater movement range and the minimal dressings facilitate nursing procedures.

c. **Closed Methods.**
   1. A layer of Tullegras is covered with an absorbent material with or without an intervening antibacterial agent. These dressings must be changed daily in order to prevent bacterial proliferation in the warm, moist atmosphere. The nursing chores are enormous, and time-consuming. The sheer bulk of dressings precludes adequate physiotherapy.

Since the task of changing the dressings is so great, some deem it worthwhile to do these dressings under general anaesthesia and also find it a good time to splint patients optimally. However, the possibilities of mobilisation of patients with minimal dressings and the ease of placing patients with minimal dressings in water baths makes me inclined to discard this particular closed method.

2. A closed method which has an enormous appeal to me is the use of disposable plastic bags or gloves, which can be readily changed after lavaging the individual twice daily. The bags are especially useful on the hands and feet, whereas adhesive Op-site is useful for wrapping around the limbs and the trunk. By this method the burnt areas are kept moist and virtually painfree, thus enabling unlimited movement from the onset.

   Also progressive thrombosis of areas underlying burns is kept to a minimum and so progressive tissue loss and underlying deep structures such as tendons and muscle may be spared from becoming implicated in the malignant necrotic process. Serum losses, however, are not prevented.

3. Homograft and porcine heterograft dressings may be used similarly to the plastic covers and these require only 48-hourly replacement and they have the added advantage of decreasing plasma losses to an absolute minimum. Costs are very high and hyperpyrexias have made us somewhat less enthusiastic to use this method.

Regardless of the type of dressing, posturing of the patient is vital to the prevention of contractures. Splinting is thus a necessary evil in view of the fact that few patients are prepared to persist with physiotherapy, when not being personally encouraged or goaded.

The **healing** of burns tends to follow the ensuing pattern:—

At the junction of the eschar and the visible tissues granulation tissue develops.

Initially fibrin is deposited and fibroblasts actively lay down procollagen which matures to collagen. Simultaneously capillary budding and canalisation occurs.

Along with these processes enzymatic debridement separates the eschar and macrophage activity removes such necrotic material.

In this "soup" bacterial proliferation may occur and thus exposes the patient to possible development of septicaemia.

Should it be that some epidermal remnants persist in the dermal layers, spontaneous epithelialisation will occur. However, it is significant that in a high percentage of these cases hypertrophic scarring and possibly Keloid formation takes place. One considers that an auto-immune reaction is responsible for this deviation from the normal healing process.

Hypertrophic scars develop within the limits of the traumatised area and usually regress within 18-24 months, leaving widened atrophic scars.

Keloids are usually not referred to until at least 24 months have elapsed and it is usual that the lesions exceed the bounds of the traumatized zone. Often keloids will outgrow their blood supply and become necrotic and ulcerated.

Surgically burns are mainly treated, after spontaneous or surgical escharectomisation, by split skin grafting using either whole sheets or skin or meshed skin.

Split skin grafts are known also as Thiersch grafts and may be thin, medium or thick. The thinner the graft the greater the chance of a good take and the speedier is the healing of the donor site. Donor site healing is spontaneous since the basement cellular layer of the epidermis remains intact.

Tangential excision whereby at a very early stage the devitalised skin is planed down to a bleeding surface and then skin grafted speeds up mobilisation and discharge with minimal immobilisation.

Rather infrequently full-thickness of Wolffe grafts are employed to give good quality supple grafts; however, the "take" of such a graft is precarious and the donor site requires either to be split-skin grafted or repaired under tension since epidermal regeneration cannot occur except from the periphery.

Where vital structures are implicated in the injury and a substantial protective coverage is necessary a pedicle flap, which retains or is ultimately separated from its pedicle, is utilised.

The dynamic process of graft healing gives some indication as to when and how vigorously mobilisation may be effected.

For the first 24-48 hours nutrients and gases nourish the graft by means of diffusion through the plasmatic layer. Commencing immediately after the application of the graft, fibrin tenuously anchors more firmly and as this pro-collagen matures within 6-21 days to collagen, the anchorage is improved. Until this maturation is far advanced, with increased hydrostatic pressure, e.g. by dependent posturing vesiculation readily occurs and every effort should be made to prevent this by means of bandages and elastic support. Rupturing of the blisters may lead to ulceration. Gentle graded movement may be commenced from the 4th day and from the 10th day vigorous mobilisation should be fairly safe.

From 6-24 hours vascular budding and canalisation occurs and proceeds vigorously for 21-28 days.

Since the new capillaries are very fragile, red cell extravasation easily occurs with modest increases in hydrostatic pressure. Local haemosiderosis, with the consequent pruritis, may be very troublesome to the patient. In order to prevent this, elastic bandaging is suggested.

Collagen as it matures tends to contract and in so doing strangles the capillaries, producing after 6-12 weeks contracted pale scars, and where this occurs over unrestrained areas such as joints, contractures with tendon, muscle and joint capsule shortening may result.

It is these contracture potentials which demand persistent vigorous, active physiotherapy, and splintage, for the time when the physiotherapist turns her back to the uninspired, unmotivated, often helpless and hopeless patient.