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Advances in Burn Management

An essay

A protocol submitted for fulfillment of master degree in general surgery

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Introduction

Management of burn patients requires a complex interaction of surgical, medical, critical care, and rehabilitation approaches. Severe burn patients are some of the most challenging critically ill patients who may have multiple-system organ failure with life-threatening complications ***(Ong et al., 2006).***

Recent changes and new therapies have been incorporated into burn care worldwide through the efforts of clinical and basic research. This essay will summarize important advances in treating burn patients ***(Bessey PQ, 2007).***

It is often difficult to determine what specific discoveries change health delivery in burns. The areas of advancement in burn care that have improved outcome are myriad and include acute care, burn wound treatment, control of the hypermetabolic responses, and control of life threatening infections ***(Jeschke et al., 2008).***

The mortality and length of hospital stay of burn patients have been greatly reduced over the last 25 years. In the 1960's, the likelihood of survival was only 50% for pediatric burns covering 35-44% of the TBSA (total body surface area), and few patients with burn sizes above 45% TBSA survived. The average length of stay for the acutely burn patient was 103 days. Today, the LB_{50} (lethal burn size for 50% of the patients) for patients exceeds 95% TBSA, and the average length of hospital stay for most serious burn injuries can be expected to be only 0.5 days per percent of TBSA that is burned ***(Celis et al., 2003).***

This is truly a remarkable achievement and is striking testimony to the concentrated effort in personnel and resources that have been directed toward severely burn patients.

Specific aspects of burn care that have dramatically improved in burn hospitals include: treatment to improve acute care, such as resuscitation, early excision and grafting, the control of infections and improving the immune system, improvements in provision of metabolic and nutritional requirements, and the evolution of effective skin banks, infection control, and alternative wound-closure materials and strategies ***(Cleland HJ, 2007).***

Most importantly however, not only have the length of stay and survival rates improved over the last 25 years, it is also a remarkable achievement that severely burned patients have tremendously improved long-term outcomes. Over the last decade major advances enabled burn patients to be effectively rehabilitated and reintegrated as productive members into society. These patients have skills and developmental improvements that are truly outstanding leading to effective, productive, and thoughtful members of society and also making this world a better place ***(Suman et al., 2003).***

CHAPTER I

INTRODUCTION TO BURN

I. Definition and facts

A **burn** is a type of thermal injury to flesh caused by heat, electricity, chemicals, light, radiation or friction. Most burns affect only the skin (epidermal tissue and dermis). Rarely, deeper tissues, such as muscle, bone, and blood vessels can also be injured. Burns may be treated with first aid, in an out-of-hospital setting, or may require more specialized treatment such as those available at specialized burn centers ***(Herndon, 2007).***

All over the world, 22 people die hourly because of burn. In 2004, nearly eleven million people worldwide were burned severely enough to require medical attention. In India, over one million people are moderately or severely burnt every year. In 2000, direct costs for care of children with burns in the United States of America exceeded US$ 211 million ***(WHO, 2012).***

II. Body response to burn

Burn injuries result in both local and systemic responses.

A. Local response

The three zones of a burn were described by ***Jackson***;

1. 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.

2. Zone of stasis*:* The surrounding zone of stasis is characterized 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 edema can convert this zone into an area of complete tissue loss.

3.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 ***(Kao and Garner, 2000).***

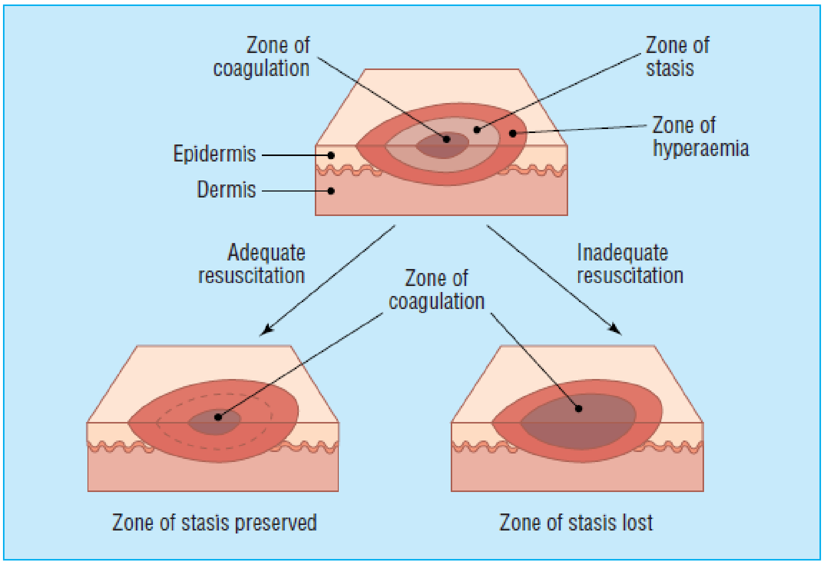

Fig. 1: Jackson's burns zones and the effects of adequate and inadequate resuscitation (Hettiaratchy and Dziewulski, 2004).

B. 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.

1. 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. These changes, coupled with fluid loss from the burn wound, result in systemic hypotension and end organ hypoperfusion.

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

3. Metabolic changes: The basal metabolic rate increases up to three times its original rate. This, coupled with splanchnic hypoperfusion, necessitates early and aggressive enteral feeding to decrease catabolism and maintain gut integrity.

4. Immunological changes: Non-specific down regulation of the immune response occurs, affecting both cell mediated and humoral pathways ***(Andronicus et al., 1998).***

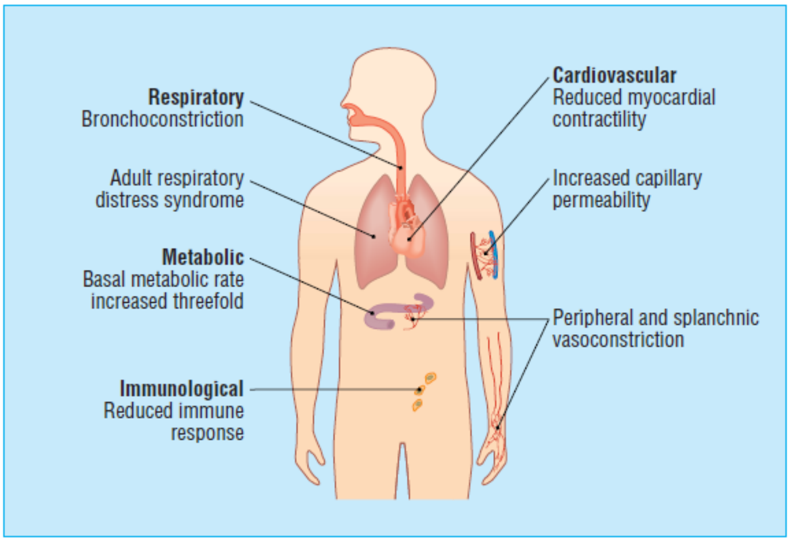

Fig. 2: Systemic changes that occur after a burn injury (Hettiaratchy et al., 2004).

III. Sign and symptoms

Burns can be classified by mechanism of injury, depth, extent and associated injuries and comorbidities.

A. By mechanism

Burns are caused by a wide variety of substances and external;

1. Flame and flash burn

Flash and flame burn injuries represent approximately half of the admissions to American regional burn centers. Explosions of natural gas, propane, gasoline, and other flammable liquids cause intense heat for a very brief time ***(Herndon, 2007).***

While such burns will generally heal without extensive skin grafting, they may cover large areas and may be associated with thermal damage to the upper airway (inhalation injury) ***(Byrom, 1984).***

2. Scald burn

Scalding (from the Latin word *calidus*, meaning hot) is caused by hot liquids (water or oil) or gases (steam) ***(Allasio and Fischer, 2005).***

3. Chemical burn

Most chemicals that cause chemical burns are strong acids or bases. Hydrofluoric acid can cause damage down to the bone and its burns are sometimes not immediately evident ***(Luterman and Curreri, 1990).***

4. Electrical burn

Burns caused by either an electric shock or an uncontrolled short circuit (a burn from a hot, electrified heating element is *not* considered an electrical burn) ***(Arnoldo et al., 2004).***

Low-voltage (<500 AC volts) injury causes no skin burns or marks. This is sufficient to cause cardiac arrest and ventricular fibrillation. High voltage electricity, on the other hand, is a common cause of third and fourth degree burns due to the extreme heat yielded by high temperature arcs and flashover associated with voltages over 1000 volts ***(Wright and Davis, 1980).***

5. Contact burn

In order to get a burn from direct contact, the object touched must either have been extremely hot or the contact was abnormally long. The latter is a more common reason, and these types of burns are commonly seen in people with epilepsy or those who misuse alcohol or drugs. They are also seen in elderly people after a loss of consciousness ***(Gibran et al., 1994).***

6. Radiation burn

They are caused by protracted exposure to UV light (as from the sun), tanning booths, radiation therapy (in people undergoing cancer therapy), sunlamps, radioactive fallout, and X-rays. Tanning booths also emit these wavelengths and may cause similar damage to the skin ***(Hirsch and Bowers, 1992).***

B. By depth:

Currently, burns are described according to the depth of injury to the dermis and are loosely classified into first, second, third, and fourth degrees. This system was devised by the French barber-surgeon Ambroise Pare and remains in use today ***(Walls et al., 2009).***

The following tables describe degrees of burn injury under this system as well as provide pictorial examples;

Names	Layers involved	Appearance	Texture	Sensation	Time to healing	Complications	Example
First degree	Epidermis	Redness (erythema)	Dry	Painful	1wk or less	None	
Second degree (superficial partial thickness)	Extends into superficial (papillary) dermis	Red with clear blister. Blanches with pressure	Moist	Painful	2-3wks	Local infection/cellulitis	
Second degree (deep partial thickness)	Extends into deep (reticular) dermis	Red-and-white with bloody blisters. Less blanching.	Moist	Painful	Weeks - may progress to third degree	Scarring, contractures (may require excision and skin grafting)	
Third degree (full thickness)	Extends through entire dermis	Stiff and white/brown	Dry, leathery	Painless	Requires excision	Scarring, contractures, amputation	
Fourth degree	Extends through skin, subcutaneous tissue and into underlying muscle and bone	Black; charred with eschar	Dry	Painless	Requires excision	Amputation, significant functional impairment, possible gangrene, and in some cases death.	

Table 1: A table classifying burn injuries according to the depth (Walls et al., 2009).

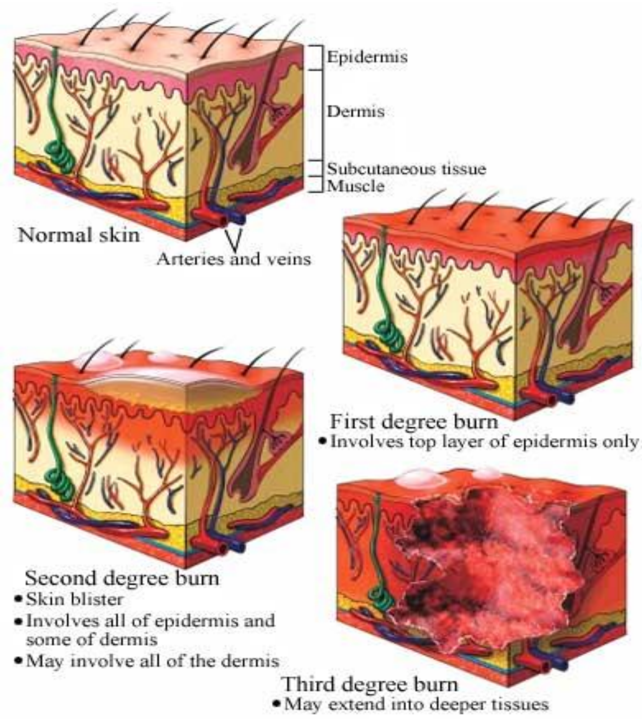

Fig. 3: burn degrees (Nucleus Medical Art, 2009)

C. By severity:

In order to determine the need for referral to a specialised burn unit, the American Burn Association devised a classification system to aid in the decision-making process. Under this system, burns can be classified as major, moderate and minor. This is assessed based on a number of factors, including total body surface area (TBSA) burnt, the involvement of specific anatomical zones, age of the person and associated injuries ***(American Burn Association, 2011).***

1. Major

Major burns are defined as:

a) Age 10-50yrs: partial thickness burns >25% of total body surface area
b) Age <10 or >50: partial thickness burns >20% of total body surface area
c) Full thickness burns >10%
d) Burns involving the hands, face, feet or perineum
e) Burns that cross major joints
f) Circumferential burns to any extremity
g) Any burn associated with inhalational injury
h) Electrical burns
i) Burns associated with fractures or other trauma
j) Burns in infants and the elderly
k) Burns in persons at high-risk of developing complications

These burns typically require referral to a specialized burn treatment center.

2. Moderate

Moderate burns are defined as:

a) Age 10-50yrs: partial thickness burns involving 15-25% of total body surface area
b) Age <10 or >50: partial thickness burns involving 10-20% of total body surface area
c) Full thickness burns involving 2-10% of total body surface area

Persons suffering these burns often need to be hospitalized for burn care.

3. Minor

Minor burns are:

a) Age 10-50yrs: partial thickness burns <15% of total body surface area
b) Age <10 or >50: partial thickness burns involving <10% of total body surface area
c) Full thickness burns <2% of total body surface area, without associated injuries

These burns usually do not require hospitalization ***(American Burn Association, 2011).***

D. By surface area:

Burns can also be assessed in terms of total body surface area (TBSA), which is the percentage affected by partial thickness or full thickness burns. First degree (erythema only, no blisters) burns are not included in this estimation. The Wallace rule of nines is used as a quick and useful way to estimate the affected TBSA. More accurate estimation can be made using Lund & Browder charts, which take into account the different proportions of body parts in adults and children. The size of a person's hand print (palm and fingers) is approximately 0.8% of their TBSA, but for quick estimates, medical personnel round this to 1%, slightly overestimating the size of the affected area ***(Ames, 2010).***

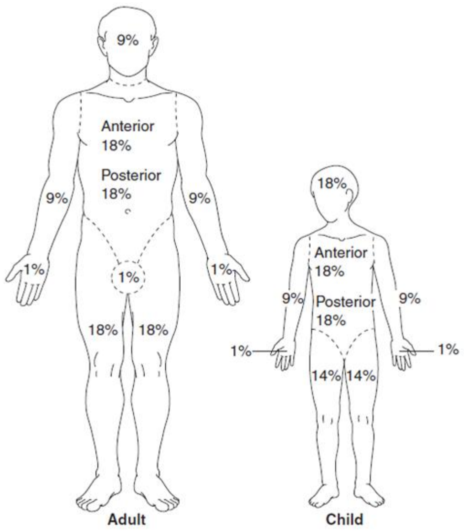

Fig. 4: A diagram illustrating the rule of nine in estimating the percentage of burn distribution (Knaysi, 1967).

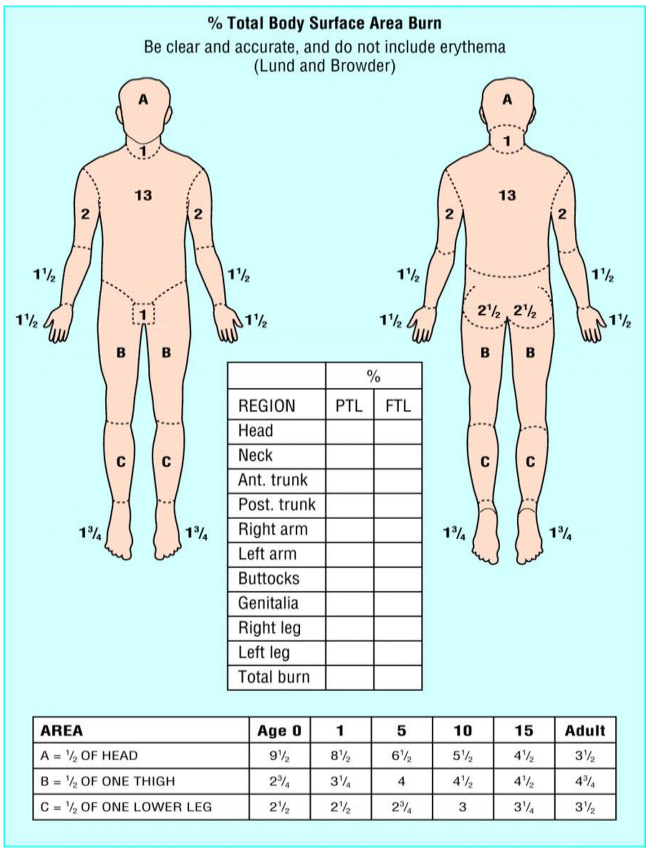

Fig. 5: A diagram showing Lund and Browder chart which estimates the percentage of burn distribution (Herndon, 2007).

IV. Management

Both general and local management are essential for burn care.

A. General management:

1. Resuscitation

The resuscitation and stabilization phase begins with the reassessment of the injured person's airway, breathing and circulatory state. Appropriate interventions should be initiated to stabilize these. This may involve targeted fluid resuscitation and, if inhalation injury is suspected, intubation and ventilation. Once the injured person is stabilized, attention is turned to the care of the burn wound itself. Until then, it is advisable to cover the burn wound with a clean and dry sheet or dressing. Early cooling reduces burn depth and pain, but care must be taken as uncontrolled cooling can result in hypothermia ***(Tintinalli and Judith, 2010).***

Children with >10% total body surface area burns, and adults with >15% total body surface area burns need formal fluid resuscitation and monitoring (blood pressure, pulse rate, temperature and urine output). Once the burning process has been stopped, the injured person should be volume resuscitated according to different resuscitation formulas ***(Herndon, 2007).***

Other fluid resuscitation formulas are less commonly used;

Adult burn resuscitation formulas.

Phase	First 24 h post burn	Second 24 h post burn
Burn Budget Formula	LR: 1000 mL–4000 mL 0.5 normal saline: 1200 mL Colloid: 7.5% of body weight Glucose in water: 1500–5000 mL	LR: 1000 mL–4000 mL 0.5 normal saline: None Colloid: 2.5% of body weight Glucose in water: 1500–5000 mL
Evans Formula	Normal saline: 1 mL/kg/% TBSA burn Colloid: 1 mL/kg/% TBSA burn D5W: 2000 mL	Normal saline: 0.5 mL/kg/% TBSA burn Colloid: 0.5 mL/kg/% TBSA burn D5W: 2000 mL
Brooke Formula	LR: 1.5 mL/kg/% TBSA burn Colloid: 0.5 mL/kg/% TBSA burn D5W: 2000 mL	LR: 0.5 mL/kg/% TBSA burn Colloid: 0.25 mL/kg/% TBSA burn D5W: 2000 mL
Modified Brooke	LR: 2 mL/kg/% TBSA burn With ½ of the volume given during the first 8 h and ½ over the next 16 h	Colloid: 0.3–0.5 mL/kg/% TBSA burn D5W: to maintain urine output
Parkland	LR: 4 mL/kg/% TBSA burn With ½ of the volume given during the first 8 h and ½ over the next 16 h	Colloid 20%–60% of calculated plasma volume D5W: to maintain urine output of 0.5 mL/kg/h in adults
Modified Parkland	LR: 4 mL/kg/% TBSA burn With ½ of the volume given during the first 8 h and ½ over the next 16 h	5% albumin at a rate of (0.3–1 mL/kg/% burn)/16 per h
Monafo Hypertonic Formula	250 mEq Na, 150 mEq Lactate, 100 mEq Cl with volume adjusted to maintain urine output of 30 mL/h	⅓ normal saline with volume adjusted by urine output

LR: Lactated Ringer's solution
TBSA: total body surface area
D5W: 5% dextrose in water

Table 2: Different fluid resuscitation formulas for adult burn patients (Warden, 1992).

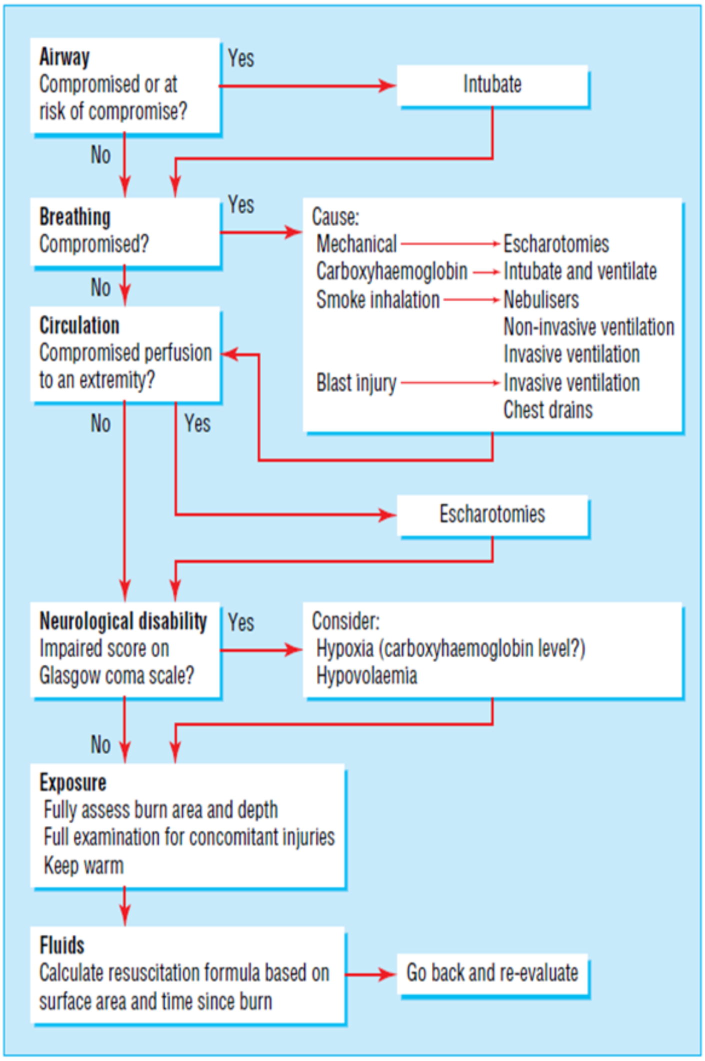

Fig. 6: Algorithm for primary survey for a major burn injury (Herndon, 2007).

2. Medical treatment

Antibiotics, analgesics, inotropic drugs, β-blockers (to decreases cardiac load), bronchodilators, expectorants (in cases of inhalation injury), drugs for prophylaxis against stress ulcer, antiemetics, GIT motility regulators, diuretics, anticoagulants (for DVT prophylaxis), antihistaminic drugs (for itching), anabolic drugs, vitamins and minerals (for compensating deficiencies) may be used in protocols for treatment of burn patients ***(Faucher and Furukawa, 2006).***

B. Local management

1. Wound care

Debridement cleaning and then dressings are important aspects of wound care. The wound should then be regularly re-evaluated until it is healed ***(Herndon, 2007).***

In the management of first and second degree burns little quality evidence exists to determine which type of dressing should be used ***(Wasiak et al., 2008).***

2. Surgery:

Circumferential burns of digits, limbs or the chest may need urgent surgical release of the burnt skin (escharotomy) to prevent problems with distal circulation or ventilation ***(Herndon, 2007).***

Superficial burn wounds should heal by regeneration within two weeks. Deeper burns will not reepithelialize spontaneously and should have epithelial cover within 3 weeks to minimize scarring. The ideal covering of a burn is a split thickness skin autograft from unburnt areas ***(Papini, 2004).***

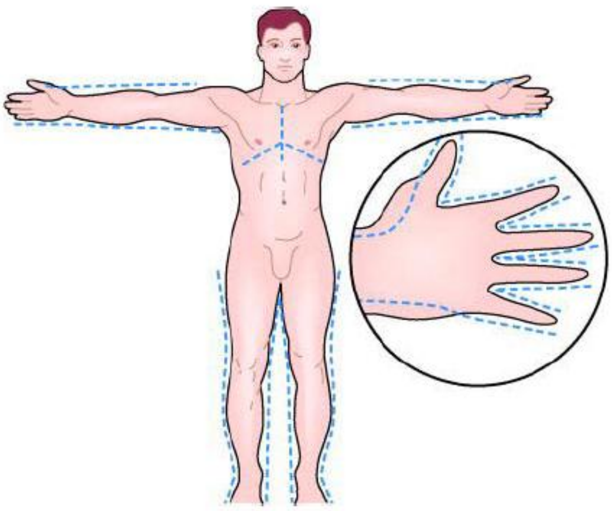

Fig. 7: Preferred sites of escharotomy incisions (Townsend et al., 2004)

V. Complications

A. Hypothermia (Judkins, 2000).

B. Compartment syndromes*: ** A life-threatening complication caused by high-volume resuscitation is abdominal compartment syndrome (ACS) ***(Berger et al., 2001).

It is defined as intra-abdominal pressure ≥20 mm Hg plus at least one new organ dysfunction. ACS has been associated with renal impairment, gut ischemia, and cardiac and pulmonary malperfusion ***(Malbrain et al., 2006).***

C. Deep venous thrombosis (Faucher and Conlon, 2007).

D. Heparin-induced thrombocytopenia (Greenhalgh et al., 2007).

E. Neutropenia (Herndon, 2007).

F. Stress ulcers (Guillamondegui et al., 2008).

G. Acute tubular necrosis: caused by myoglobin and hemoglobin released from damaged muscles and red blood cells, when adequate fluid resuscitation has not been achieved ***(Holm, 2000).***

H. Adrenal insufficiency: Those with massive burns have higher cortisol levels but may be resistant to serum cortisol increases in response to stimulation ***(Fuchs et al., 2007).***

I. Infection/inflammation/sepsis: linked to impaired resistance from disruption of the skin's mechanical integrity and generalized immune suppression ***(Greenhalgh et al., 2007).***

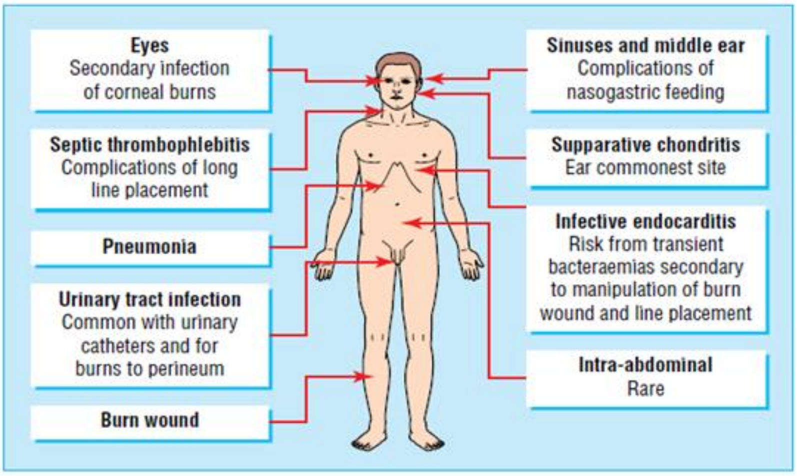

Fig. 8: Possible sources of infection in burn patient (Herndon, 2007).

CHAPTER II

BURN PROGNOSTICS

The progress that has been made in burn care over the past four decades has dramatically increased survival rates for burn victims. The advances that led to this success accompanied the following three giant steps in the history of modem burn care; the introduction of topical antibiotic therapy into the wound care armamentarium, the adoption of the technique of excising the burn eschar of large burns and improvements in the technology and clinical skill in the management of burn wounds ***(Herndon, 2007).***

Single factors, scoring systems and biomarkers can be used as prognostics for burn injuries mortality against survival;

I. Single factors:

A. Personal history of the patient:

1. Age

Bull and Squire in their pioneering paper in ***1949*** were able to state that age has a very marked effect on the outcome of a burn ***(Roi et al., 1983).***

In ***2005, Moreau et al*** have shown that curvilinear effect of age on mortality in burn patients was the least at age 21.

Higher mortality rates are found in older patients especially over 70 can be explained by higher possibility of preexisting medical comorbidities, low immunity, higher susceptibility to infection ***(Le et al., 1986).***

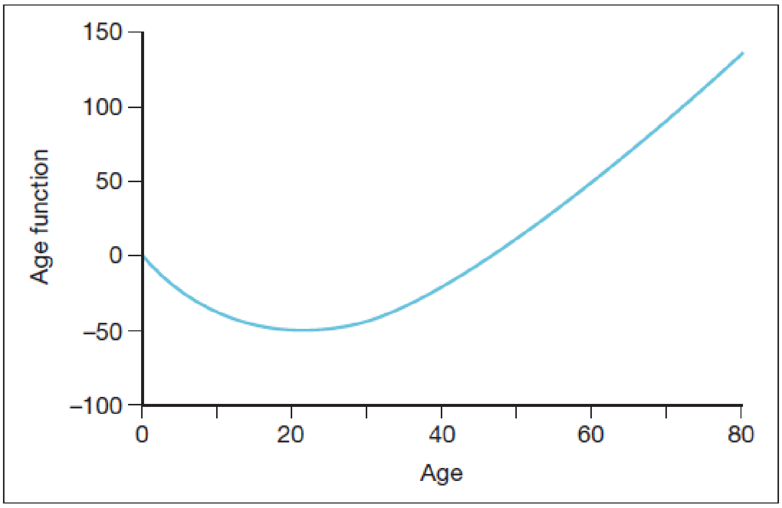

Fig. 9: Effect of age on mortality. Effect is minimal at age 21 (Herndon, 2007).

2. Sex

Most studies that have included an analysis of the influence of sex on mortality have reported a positive correlation, mortality being higher in females ***(George et al., 2005).***