

Burn Management In I.C.U.

An Essay Submitted for Partial Fulfillment of the Master Degree in Intensive Care

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Introduction

Burn injuries are considered as a major challenge for intensivists and remain as a leading cause of mortality and long term dysfunction in spite of significant medical advances and improvement in over all mortality following burn injuries (*Pavoni et al., 2010*).

Burn patients are considered as a heterogenous population with wide variation in age, mechanism of injury, depth, and site of burn and a different comorbidity (*Pavoni et al., 2010*).

Burn injuries are associated with autonomic, physiologic and immunologic alterations which require special care. Cutaneous injury results in significant fluid loss as well as the release of multiple inflammatory mediators when disseminated by circulation to central organ,Bacteria and inflammatory mediators can cause cardiovascular compromise, a breakdown of gastrointestinal mucosal integrity and multiple organ failure. Inhalational injury can further accelerate these responses (*Khan, 2006*).

Burn wound infections are considered as one of most important and potentially serious complications that occur within acute period following injury. Advances in medical care have changed the principal cause of death in burn patients from burn shock to wound sepsis. The development of topical and systemic antimicrobial agents, improved nutritional support for the hypermetabolic response and the use of surgical techniques for early wound excision have now changed the primary causes of death from wound sepsis to pulmonary sepsis which often follows an inhalational injury (*Ralfa, 2011*).

Introduction and Aim of The Work

The physiological recovery of burned patient should be viewed as a continuing process with the patient slowly moving towards ultimate physical recovery and social reintegration. Recovery from thermal injury can be divided into 3 phases: resusitation phase, acute rehabilitation and long term rehabilitation (*Rutan et al., 2005*).

Many advances have been made in burn management and these advances have resulted in marked decrease in mortality rates. The major advances have been in two areas. The first is in the rapid and safe removal of burn tissues before the onset of infection and wound closure. The second advance has been in the area of critical care management which allows the patient to avoid major organ failure especially pulmonary failure (*Demling*, 2006).

To provide comprehensive care for severely burned patients, close collaboration is required among members of multidisciplinary team. This burn team comprises critical care physicians, plastic surgery physicians, nurses, physical therapists, occupational therapists, nutritionists, psychologists, social mothers and family support members (*Demling*, 2006).

Aim of The Work

The aim of this essay is to discuss the updates and new modalities of management of burned patient in critical care units especially in fluid resuscitation, electrolyte homeostasis and protection against bacterial infection.

Also how to prevent early cardiopulmonary complications, how to minimize patient's pain and discomfort.

Finally the discharge plan by the entire staff of burn team to ensure that the patient can care for himself after discharge.

Skin Anatomy ad Physiology

Human skin is the largest organ of the body and covers an area in excess of $2m^2$ with a mass representing approximately one sixth of total body weight and up to one third of the circulating blood volume can be supplied to the skin with a basal flow of approximately 300 ml/min (*Holm et al., 2004*).

Human skin is the interface between the human internal and external environment. A multifunctional organ provides protection, sensation, thermoregulation, biochemical and metabolic functions. Disruption of the skin through exposure to mechanical or traumatic means can lead to increased fluid loss, infection, hypothermia, scarring, increased or decreased sensitivity, compromised immunity, change in body image and invasion by squamous cell carcinoma. Systemic diseases such as diabetes, aging and genetic alterations also can lead to delayed healing and impaired functioning of this organ (*Mark et al., 2002*).



I) Skin Anatomy:-

Chapter One

The Skin is made up of the following layers with each layer performing specific function (*Briggamann., 2002*).

a) Epidermis :

The epidermis is the thin outer layer of the skin, is derived from the embryonic ectoderm and consists of five layers from the top to the bottom :

- Stratum coreum.
- Stratum lucidum.
- Stratum granulasum.
- Stratum spinosum.
- Stratum besale.

Stratum corneum (horny layer) : This layer consists of fully mature keratinocytes which contain fibrous proteins (keratins). The outermost layer is continuously shed. The stratum corneum prevents the entry of most foreign substances as well as the loss of fluid from the body.

The basal layer : The basal layer is the deepest layer of the epidermis, containing basal cells. Basal cells continually divide, forming new keratinocytes that replace the cells that are shed from the skin's surface.

The epidermis also contains melanocytes which are cells that produce melanin (skin pigment).

b) Dermis :

The dermis is the middle layer of the skin. The dermis contains the following:

- 1- Blood vessels.
- 2- Lymph vessels.
- 3- Hair follicles.
- 4- Sweat glands.
- 5- Collagen bundles.
- 6- Fibroblasts.
- 7- Nerves.

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The dermis is held together by a protein called collagen, made by fibroblasts. This layer gives skin flexibility and

strength. It also contains pain and touch receptors.

c) Subcutaneous fat layer :

The Subcutaneous fat layer is the deepest layer of skin. It consists of a network of collagen and fat cells, helps conserve the body's heat and protects the body from injury by acting as a shock absorber.

Other cells found in the dermis are macrophages or wandering histocytes, mast cells and lymphocytes. Mast cells are often seen near blood vessels just below the epidermal-dermal junction. Mast cells have binding sites for immunoglobulin E (*Briggamann, 2002*).

II) Skin Physiology:-

Knowledge of skin function is as important as knowledge of skin anatomy.

1. Protection:

Protection from the environment is one of the most fundamental functions of skin. It protects the rest of the body from chemicals, bacteria, viruses, aqueous liquids, mechanical trauma and ultraviolet radiation (*Auerbach, 2001*).

Skin acts as a barrier to protect internal organs from exposure to the outside environment and maintains an internal homeostatic environment. It is an effective barrier against the excessive loss of fluids that can occur in.a burn injury. A person with a 30 % body burn may lose approximately 4L of fluid compared to 710 ml in a healthy adult with intact skin *(Auerbach, 2001).*

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Intact stratum corneum of the dermis is an effective pathogenic barrier for bacteria and viruses. The dead keratinized cells or squames cells which are shed with daily bathing. The lipid coating of the skin surface is deposited by sebaceous glands and provides an acidic coating with a pH ranging from 4 to 6.8, with a mean of 5.5, which effectively inhibits infection. Sebum also has many antibacterial effects (*Guyton, 2002*).

Skin pigmentation provides protection against ultraviolet (UV) radiation. The most harmful spectrum is UV-A, which is the long waveform that has a spectral range from 320 to 400 nm (*Hardwicke et al.,2008*)

2. Thermoregulation:

Another important function of the skin is the maintenance of an internal homeostatic thermal environment to ensure the continued function of other major organ systems. Circulation and sweating are two of the primary thermoregulatory mechanisms. In warm environments, there is increased blood flow and sweating so heat loss through conduction, convection, radiation and evaporation dissipate the heat and the reverse in cold environments (*Guyton, 2002*).

3. Sensation:

The skin has an elegant system of nerve receptors to sense pain, touch, pressure, vibration and temperature. Nerve receptors in the epidermis and dermis transmit impulses to the cerebral cortex for interpretation. Burning and itching are considered a combination of the four basic sensations which are pain, pressure, vibration and temperature. (*Goldsmith, 2001*).

4. Metabolism and Biochemistry:

Vitamin D is synthesized in the skin in the presence of sunlight. Vitamin D is important in the mineralization of bone and in calcium and phosphate metabolism. Adipose tissue, which constitutes a major energy store, is found subcutaneously in the hypodermis (*Patel et al., 2002*).

5. Immunology:

Cells of skin immune system provide protection from invading microorganisms. Among these cells are the Langerhans cells, an antigen-presenting cell found in the epidermis, tissue macrophages, which can engulf, phagocytose and then destroy bacteria and mast cells which contain histamine and induce an inflammatory response. T lymphocytes and dendritic cells are the adaptive cells of the skin immune system that are resident in skin (*Briggamann,* 2002).

Pathophysiological Changes in Burn Injuries

Types of Burns:

A- According depth of the burn.

- B- According to the causes.
- C- According to the severity

A- According depth of the burn:

<u>First-degree burns</u> are usually limited to redness (erythema), a white plaque and minor pain at the site of injury. These burns usually extend only into the epidermis

<u>Second-degree burns</u> additionally fill with clear fluid, have superficial blistering of the skin and can involve more or less pain depending on the level of nerve involvement. Second-degree burns involve the superficial (papillary) dermis and may also involve the deep (reticular) dermis layer.

<u>Third-degree burns</u> additionally have charring of the skin and produce hard, leather-like eschars that has been separated from the unaffected part of the body. Frequently, There is also purple fluid. These types of burns are often painless because nerve endings have been destroyed in the involved areas.

<u>Fourth-degree burns</u>: Burns that injure the tissues underlying the skin, such as the muscles or bones.

These burns are additionally broken down into three additional degrees: fourth-degree burns result in the skin being irretrievably lost, fifth-degree burns result in muscle being irretrievably lost and sixth-degree burns result in bone being charred (*Rosen's*, 2009).

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Degree	Anatomic correlate	Schematic aspect	Clinical aspect
I	Reddening, swelling, pain (epidermis)	No Contraction	a day a
lla	Reddening, blistering, pain (superficial dermis)		Pott
lib	Pallor, blister, pain (partial dermis)	B & B &	
ш	Greyish white or black necrosis, analgesia (complete dermis)		
IV	Carbonization (may extend to the bones and joints)		

Figure (2): The classification of burn injuries (*Timo et al., 2009*).

A newer classification of "Superficial Thickness", "Partial Thickness" (which is divided into superficial and deep categories) and "Full Thickness" relates more precisely to the epidermis, dermis and subcutaneous layers of skin and is used to guide treatment and predict outcome (*Kumaraswamy*, 2008).

Chapter Two Pathophysiological changes in burn injuries

<u>A description of the traditional and current</u> <u>classifications of burns</u>

Table (1): Description of the traditional and currentclassifications of burns.

Nomenclature	Traditional Nomenclature	Depth	Clinical findings
Superficial thickness	First – degree	Epidermis involvement	Erythema, minor pain, lack of blisters
Partial thickness superficial	Second – degree	Superficial (papillary) dermis	Blisters clear fluid and pain
Partial thickness deep	Second – degree	Deep (reticular) dermis	Whiter appearances, With decreased pain, Difficult to distinguish from full thickness
Full thickness	Third – or fourth degree	Dermis and underlying tissue and possinly fascia bone or muscle	Hard leather- like eschar, purple fluid no sensation (insensate)

(Kumaraswamy, 2008)

B- According to the causes.

1- Accidental burns:

a-Thermal injuries:-

The destruction of tissues is continuous as long as the heat source is applied and ceases shortly after removal of the heat source. Damage, as a result of thermal injury, rarely occurs below 45°C. Between 45°C and 50°C, different gradations of

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cell injury may occur and above 50°C denaturation of the protein elements of the cell become apparent (*Rosen's*, 2009).

b-Electrical Injury:

High-Voltage Electric Injury:

Electricity exerts its tissue damaging effects by conversion to thermal injury. The electrical resistance of the skin can vary dramatically depending on its moisture, cleanliness and thickness. The average resistance measurements for skin are 400.000 ohms (*Kumaraswamy*, 2008).

Injuries are divided into high-and low-voltage injuries. Low-voltage injury is similar to thermal injury without transmission to the deeper tissues. Tissue damage following a high voltage electrical injury occurs not only at the cutaneous contact point, but may also involve the underlying tissue and organs along the route taken by the current between its entrance and exit sites. The intensity of the current passing through the various organs is indirectly related to the resistance of the tissues.

Nerves, blood and muscles appear to offer the least resistance to electric current and thus sustain the maximum amount of tissue damage. As a result, misleading small cutaneous lesions may overlie extensive areas of devitalized muscle, which may liberate significant amounts of myoglobin and cause acute renal failure (*Alex et al.,2006*).

c- Chemical Injury:

There are several chemical agents, which have harmful effects on tissues:

(1) Hydrofluoric acid: an occupational hazard of glass workers and workers in certain chemical processing plants.

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(2) Phenol.

- (3) Dry alkali powders: cement (calcium oxide) burns are alkali in nature, the critical substance responsible for the skin damage is the dydroxyl ion.
- (4) Anhydrous ammonia: the ignition products of white phosphorus and the vapours of strong acids, mustard gas and chlorine can all cause significant inhalation injury *(Carsin et al., 2004).*

Injuries exert their deleterious effects of tissues by the release of thermal energy during the reaction of a strong acid or base with tissue components. The severity of tissue damage, secondary to contact with chemicals, is related to the concentration of the chemical agents, the amount of agent in contact with the tissues and the duration of contact. Chemical agents last a long time, during which destruction continues, they cause progressive damage until inactivated by reaction with tissues. (*Alex et al.,2006*).

d- Radiation Burns:

Radiation either in the form of sunlight or ionizing radiation (e.g. that used in oncologic therapy) may also cause burns.Burns that result from radiotherapy are generally sharply demarcated but may involve damaging to deeper tissues in the skin (*Kumaraswamy 2008*).

2- Iatrogenic Burns:

a- Diathermy:

Diathermy produces intense heat at the tip of the small electrode which the surgeon uses in the wound, while only slight warmth is developed in the area of the "earth-plate" give current of 350 KHz (for coagulation) and 450 KHz (for cutting) (*Timo et al.,2009*).

Chapter Two Pathophysiological changes in burn injuries

- How does diathermy cause burn in-patients undergoing surgical procedures?

- 1- The earth-plate is the plate that is placed under the bare buttocks.
- 2. A broken earth-plate may cause the patient to earth himself to any point on the operating table, thus inducing a burn under anaesthesia.
- 3. A sparing earth-plate may ignite a nearby skin.
- 4. Accidental misplacement of the diathermy electrode either on a wrong point on the patient, on the surgeon or on the assistant.
- 5. Use of diathermy in the oral cavity may cause the tracheal tube to ignite (*Williams and Phillips 2003*)

- Safety precautions against diathermy burns:

- 1- The patient should be properly and efficiently earthed.
- 2- The current between the electrode and the earth should not be more than10 HA (for ECG, etc.).
- 3- The diathermy should make an audible noise when activated and the electrode sheathed when not in use.
- 4- Earth-free, solid-state diathermy sets or battery-powered sets are preferred (*Williams and Phillips, 2003*).

b- Electrocution:

The direct current supplies (DC current), with an open current flowing into the skin can cause destruction of the tissues, resulting in a "punched- out" open sore (*Alex et al.,2006*).

c- Medical Laser:

All surgical lasers present potential hazards, both for patients and for the operating room personnel.Patients should have their eyes and skin properly protected from direct contact with the laser beam. All combustible materials and flammable anaesthetics should be kept away from the area where the laser