MANAGEMETN OF BURN INJURIES : AN UPDATE

ESSAY

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بسم الله الرحمن الرحيم

"قالوا سبحانك لا علم لنا إلا ما علمتنا
إنك أنت العليم الحكيم"

صدق الله العظيم

(سورة البقرة : الآية ٣٢)

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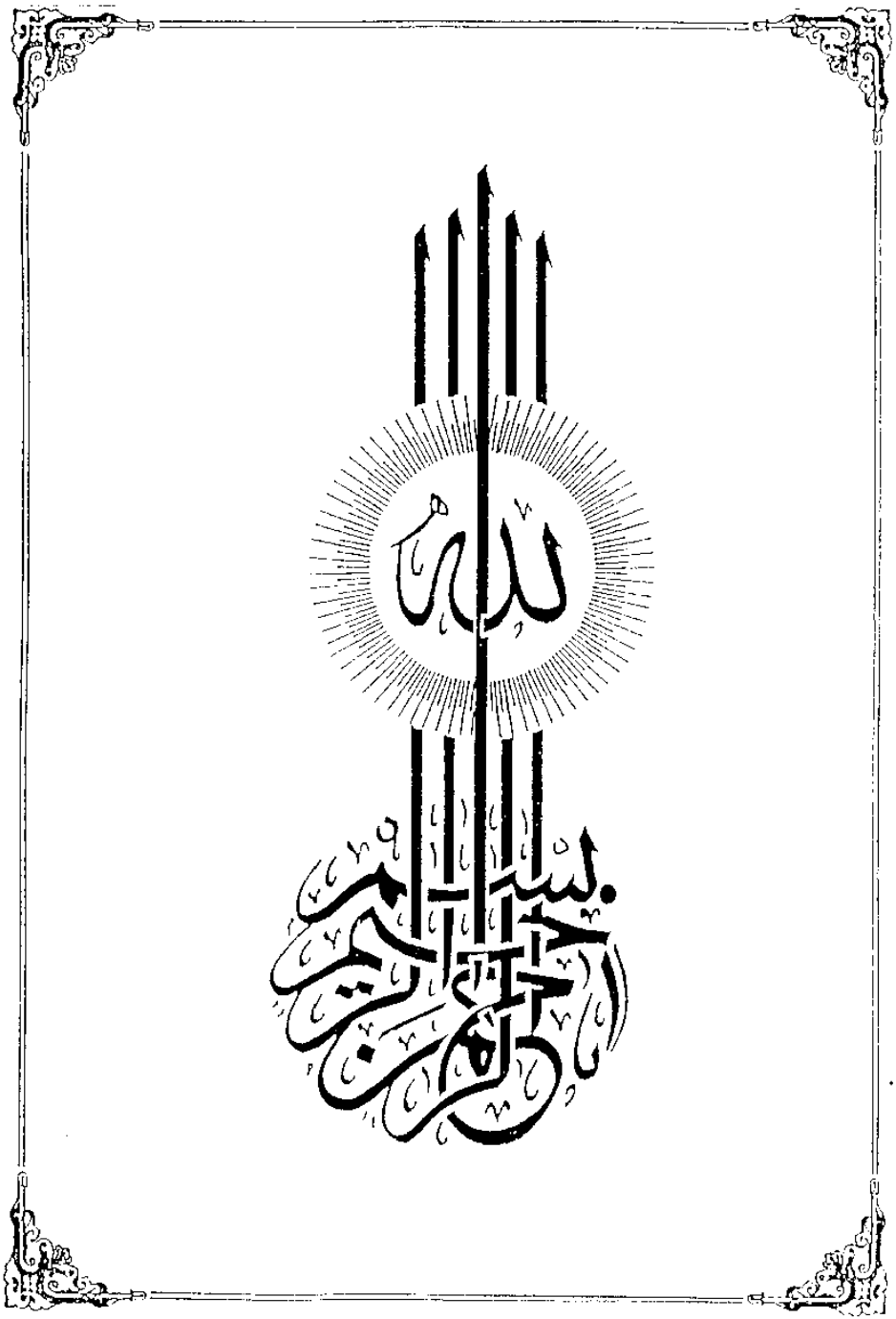

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Dedication

To
My Parents

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Introduction

Burn injuries can be one of the most serious and devastating forms of trauma that man can sustain . It continues to pose a significant problem in its occurrence and treatment .

Although it is likely that the general principles of successful treatment of this injury have been formulated , there still remain , however , many problems for the burn surgeon and the clinical team to solve in the difficult task of treating the burned patient, ***(Rossi & shakespeare , 1991)***

The multisystem effects of an extensive burn injury necessitate multispeciality medical care, intensive nursing care, and high - volume laboratory support for these patients , who are best cared for in a specialized treatment facility ***(Pruitt & Goodwin , 1987)***.

Progress in the management of burn wounds has been advancing rapidly and has contributed to the increase in survival in major body surface area burns . Topical antimicrobial therapy and early excision and skin grafting revolutionized burn wound management in the 1960s and 1970s. Early wound coverage with the newer techniques leading to normalization of systemic derangements should ultimately lead to improved survival . In the case of massive burns, these new techniques

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can lead to survival with closed wounds, which was previously technically imposible *(Wong & Munster, 1993)*.

The aim of this work is to discuss the up to date mangement of burn injuries and the various methods of treating burn wounds .

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REVIEW OF LITERATURE

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PATHOPHYSIOLOGY OF BURN INJURIES

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LOCAL EVENTS OF BURN INJURY

Local tissue injury

The degree of cell injury caused by thermal energy is determined by the temperature and the duration of the heat exposure. A heat source of less than 45°C "133°F" causes no burn even with prolonged exposure. Heat sufficient to cause protein coagulation and cell death, if applied for sufficient time, will produce full- thickness necrosis of the skin, whereas application of less thermal energy will produce only partial- thickness injury with variable damage to cells, from which they may recover *(Pruitt & Goodwin, 1987).*

The microscopic pathologic feature of the burn wound is principally coagulation necrosis. Beneath any obviously charred tissue there are 3 distinct zones. The first is the zone of "coagulation" with irriversible vessel coagulation and no capillary blood flow. Surrounding this is a zone of "stasis" characterized by sluggish capillary blood flow. Although damaged, the tissues has not been coagulated. Stasis can occur early or late. Avoiding additional injury from rubbing or dehydration may prevent stasis changes from developing and thereby prevent extension of the depth of the burn. Prevention of venous occlusion is important because it may lead to thrombosis or infarction in this zone. The third zone is that of "hyperemia" which is the usual inflammatory response of healthy tissue to non lethal injury *(Demling& Way, 1991).*

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Thermal injury is known to induce severe acute changes in living tissues. Visible swelling of the skin, blister formation and loss of surface protecting epithelium leaving wet and weeping surfaces are among the typical clinical features. With these shifts and losses of fluid from the circulation, hypovolemia develops. Without fluid substitution, major burn injuries result in hypovolemic shock. Adequate fluid resuscitation may, however, markedly aggravate the edema formation *(Lund et al., 1992).*

Oedema formation :-

Following experimental thermal injury, an early and rapid increase in tissue volume and water content has been demonstrated *(Onarheim et al., 1989).*

The highest rates of edema formation have been reported by Leape who found 80% increase in water content as early as 10 minutes postinjury *(Leape, 1986).* Demling followed the course of tissue swelling during the first week after an experimental burn injury in sheep. Edema was maximal around 6 hours postinjury, started to resolve after 24 hours, and was completely resolved in 6-7 days. This resembles the course of edema "and weight gain" in burn patients *(Demling et al., 1978).*

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It has been well broven that thermal injury produces major fluid loss from a variety of sources. The schema proposed in figure(1) demonstrates the basis of the fluid loss with the first 24 to 36 hours after injury. Thermal injury to skin eliminates its homeostatic role of water preservation for the body. Increased evaporative loss then occurs along with the coincident loss of fluid into the subepidermal bullae of second degree burns. Associated with this is a breakdown of cell membrane energy dependent pumping mechanisms that allows sodium ions into the cytosol of partially damaged cells. Water then can follow the abnormal osmotic gradient to fill up the intracellular space, resulting in cellular edema. This process occurs most extensively in fat and muscles below the frankly burned skin and probably results from thermal injury below the threshold required for cell death ***(Wachtel & Fortune, 1984)***. Initially after the thermal injury, the greatest source of fluid loss is into the so called "third space". While gut dysfunction and ileus as well as the accumulation of pleural and ascitic fluid account for some of the third space fluid, most of it is lost into the inaccessible interstitial space through the mechanism of profound abnormal capillary permeability or "sieving" associated with this type of injury ***(Moncrief, 1979)***.

Current evidence reveals that the capillary defect excists not only locally in burned tissues but extensively throughout the body in nonburned areas as well in patient with burns larger than 25% ***(Arturson, 1961)***. Not only is fluid lost through the capillary wall but

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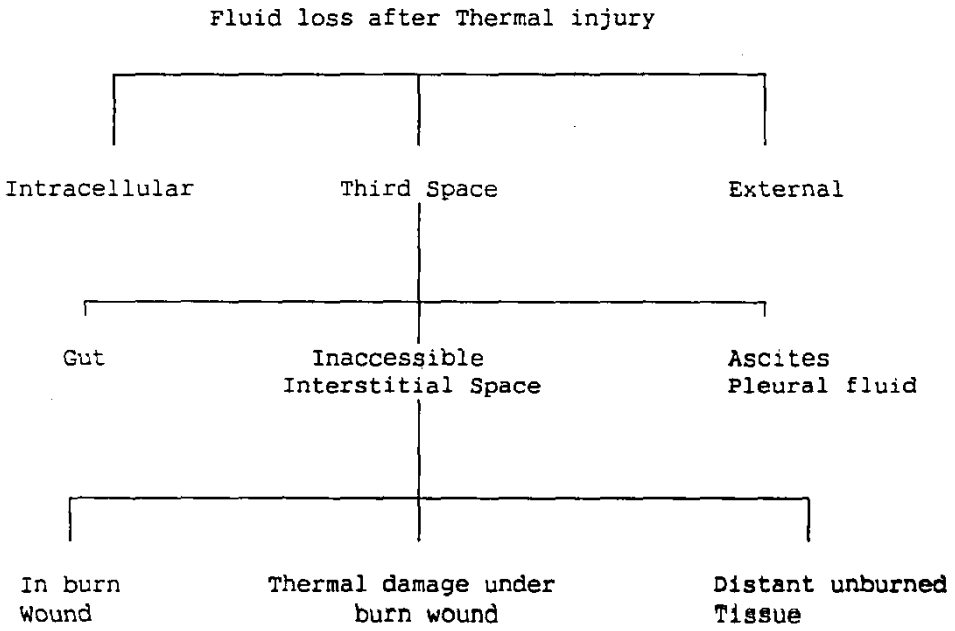

Figure 1 Schema for The initial fluid loss post burn

Source : Wachtel , T.L. and Frank , D.H.: Burns of the head and neck . W.B. Saunders company , 1984 Page 25

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