MANAGEMENT OF BURNS

E S S A Y

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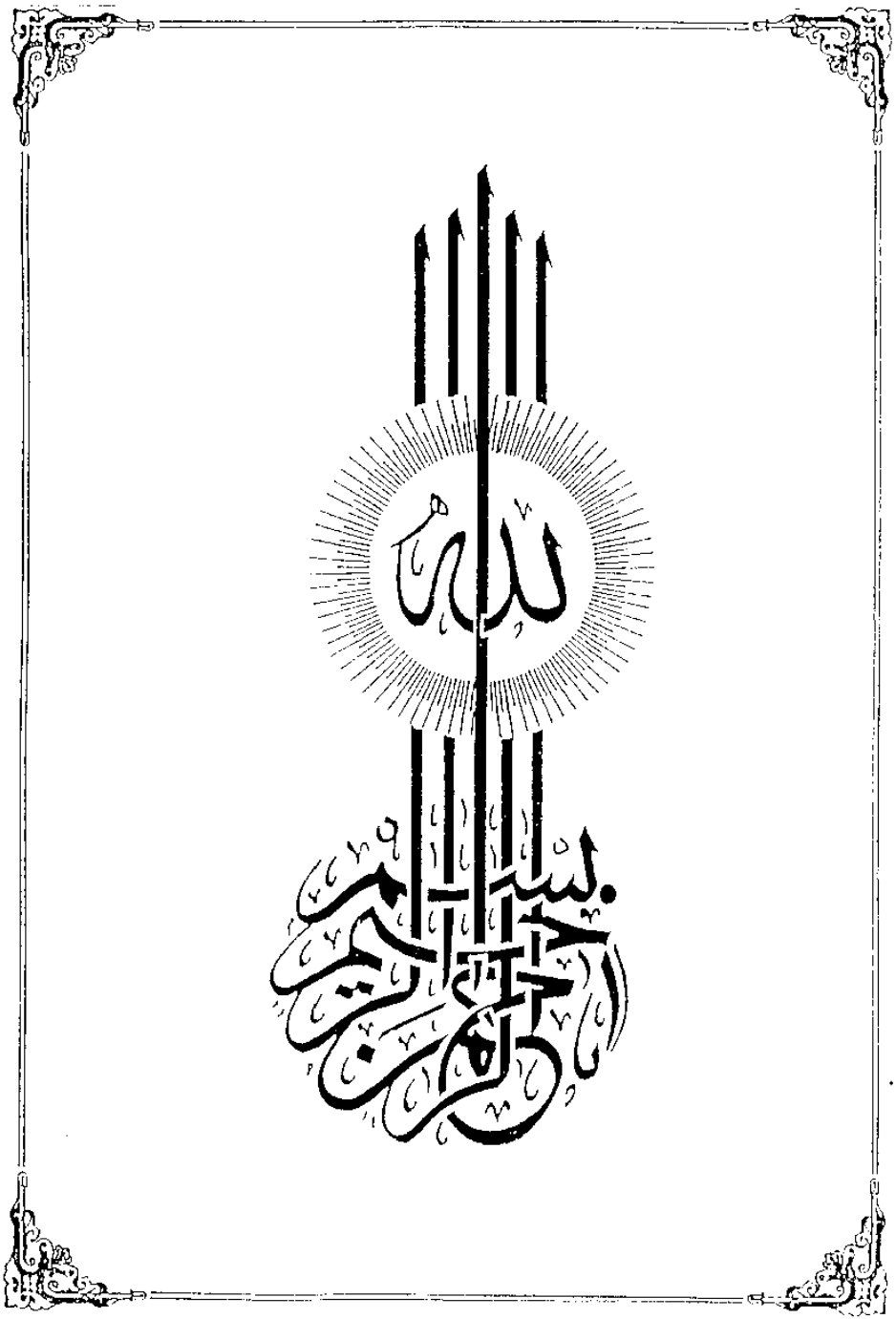

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C O N T E N T S

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INTRODUCTION

I N T R O D U C T I O N

The decreases in mortality is the expected results of an advance in the treatment of severe ilness like burns. Most of the reports suggest that mortality fell progressively until the mid nineteen-forties, since that time it has fallen little. But the survival time of patients who die from burns has increased significantly (Phillips and Cope, 1960). It appears that many patients with extensive burns, who would formerely have died in the first 48 hours from shock, today survive this phase of the illness, but fall prey subsequently to infection, metabolic disturbances or other complications for which treatment is less effective.

Recent research has improved our understanding of these complications, and has in many ways increased the effectiveness of treatment, especially for burns of moderate severity; the continued failure to reduce mortality from very severe burns has led to a revival of interest in the possible role of toxins from burned tissue and protective antibodies in the serum of convalescent patients.

The present study is concerned with management of burns which is based on the pathophysiologic changes knowledge.

The importance of general treatment is the prevention and treatment of diffirent forms of burn shock as soon as possible which is a very important problem.

The aim of the local treatment is to prevention of infection and rapid closure of the wound for cosmetic appearance and preservation of full function as far as possible.

Review Of Literature

PART I

BURNS

DEFINITION OF BURNS:

A burn is defined as the coagulative destruction of the skin and deeper tissues due to application to a surface a variety of agents, exessive heat, certain chemicals, and electrical agents. The resultent damage will depend on the temperature, strength of agents or duration of exposure which leads to not only local destruction but also wide systemic effects.

AETIOLOGICAL CLASSIFICATION OF BURNS

Burns can be classified according to aetiology into physical or chemical agents. The physical agents may be heat, irradiation or electricity and chemical agents may be acids, caustics or corrosives.

I-PHYSICAL AGENTS

The most common physical agents which cause thermal injury are:-

A-Heat exposure.

Many types of burn injury occur with heat exposure. These injuries may be solar(sunburn), flame burns, scald burns, or burns due to heat contact to a hot surface.

1)Solar(sunburn). It is the result of a complex photochemical reactions caused by exposure to sunlight or ultraviolet lamps. The burn is superficial and usually not biologically significant.

2)Flame burns. Flame causes the greatest number of significant

burns as it leads to wide local and systemic changes and mortality rate is high.

3)Scald burns. They are common in children and usually result from boiling water or hot fluids. They have the same mortality rate as those with flame burns of the same extent.

4)Heat contact to a hot surface. Exept for immersion in molten metals, burns from heat contact are usually limitted in the extent and therefore are not an important cause of death. However the burns are usually deep and often require skin grafting(Hartford and Boyed 1976).

B-Irradiation burn injury.

In addition to direct application of heat to the body surface radiation energy can produce a major thermal injury, such can occur primarily from electromagnetic spectrum which include a wide variety of radiant energy forms to which individuals are exposed almost constantly. In addition to the commonly known X-rays, infra red, ultra violet, visible light and gamma rays are also included. Radio and T.V waves are included too in this spectrum but are of no immediate significance(Moncrief 1979a).

The irradiation injury takes two forms, acute and chronic.

1)Acute irradiation injury.

It occurs due to single exposure to X or gamma radiation.

After exposure of the skin the radiation doses which do not destroy the epidermis completely, but the exposed skin areas will show a redness the so called erythema which preceeds in waves. Repetition of large doses of irradiation with a full erythema or even small doses may lead to irriversible skin changes. With large doses of irradiation, radiodermatitis bullae may occur, and sometimes it is very severe and the so called REONTIGEN ULCER develops. This takes place about sex weeks after a single large exposure. The ulcer does not heal and becomes malignant and leads finally to death(Cole and Elman 1959) or it may remain stationary for months or even years and the spontaneous healing with severely deformed scar tissue may take placr(Artz 1966).

2)Chronic irradiation injury.

Chronic exposure of the skin to ionizing radiation lead to dermatitis and malignancy of the skin. The first indication for dermatitis is changes in the skin like hypertrophy, hyperkeratosis, nails become cracked and brittle and eventually the skin becomes hyperethetic. Areas of atrophy may alternate with those of hypertrophy showing marked acanthosis and hyperkeratosis which may be of tremendous extent. Necrosis often follow incision if applied in the irradiated skin and necessitates grafting or exision with closure by full thickness skin flaps.

C-Electrical injury.

Electrical burns usually occur by a direct contact of the body with a source of electricity. The effects may be the result of electricity or heat or both as when the element of an electric fire is touched. Heat is also generated if flash occurs and if flammable objects catch fire(Prctor and London 1977). The determining factors in the severity of electrical injury are the resistance of the involved tissue, type of current, pathway of current and duration of electrical contact.

Necrosis generated by passage of electric current is proportional to the tissue resistance which with the transmission of current generate heat. Bone is the most resistant tissue to the passage of current and generates more heat than less resistant tissue(McManus 1979). Thus the tissue located in or near the centre of a limb may be injured while more superficial tissues remain viable.

The electrical tissue damage is usually characterized by two wounds, inlet and exit. High voltage electric injuries are very decptive since they often destroy deep tissues in areas where overlying skin appears intact. The limitted cutaneous involvement and in parent deep tissues in under estimation of fluid needs and the dexelopment of oliguria necessitating administration of more than estimated resuscitation fluid volume(Di Vincenti et al 1969).

II-CHEMICAL BURNS

In chemical burns, the damage of tissue occurs in a variety way. Coagulation of proteins by reduction oxidation, salt formation, corrosion, protoplasmic poisoning, metabolic inhibition, desiccation, or ischemia.

Unlike thermal injury, chemical burns tend to be progressive as long as the active agent remains in contact with the tissue. Tissue destruction ceases only when the offending agent is removed or when it has been neutralized through combination with endogenous tissue products.

Certain chemical agents can cause systemic toxicity. Tannic acid and phosphorous way cause hepatic necrosis. Picric acid and phosphorous have been associated with renal damage. Ammonia and strong acids vapours, if inhaled can cause particularly severe inhalation injury.

Chemical burn injury is important in special parts of the body. Occular lesion is accompanied with blepharospasm, excessive tearing and uncotrollable rubbing of the eyes. On progressing thereis clouding of the superficial ophthalmic tissues and progressing to fixation of the pupil, corneal ulceration, corneal perforation, iritis and lense damage. Chemical burn injury to oesophagus if progressive can lead to oesophageal stricture(McDougal et al 1978).

CLASSIFICATION OF BURNS

ACCORDING TO THE DEPTH OF INJURY

The depth of injury determine the rate of healing and subsequent cosmetic results.

DUPUYTREN'S CLASSIFICATION

Formerly, burns were classified into 6 degrees (Dupuytren's classification) indicating increase order and depth of tissue destruction.

First degree: Simple erythema due to local vasodilatation.

Second degree: Formation of blisters filled with serum.

Third degree: Coagulation of the epidermis, leaving viable remnants of hair follicles, sebaceous and sweat glands.

Forth degree: Destruction of the whole skin with loss of all epidermal elements and exposure to subcutaneous fat.

Fifth degree: Muscles and deeper structures (nerves, vessels and viscera) are involved.

Sixth degree: Bone is burnt and the limb may be charred.

CLASSIFICATION INTO THREE DEGREES

Burns are then classified into three degrees instead of six which is still present on some centres untill now.

First dgree burns:

They involve only the epidermis and characterized by erythema and minor microscopic changes. The tissue damage