THE EPIDEMIOLOGY

AND

PATHOLOGY OF BURNS

BY

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PREFACE

The study described in this dissertation was carried out in the Department of Forensic Medicine, University of Natal, under the supervision of Professor J.B.C. Botha.

This study represents original work by the author and has not been submitted in any form to another university. Where use was made of the work of others it has been duly acknowledged in the text.

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Finally, I wish to thank the members of my family, especially my wife Shakirah, for her invaluable assistance, keen interest, patience and moral support during my involvement in this project.

ABSTRACT

Burn injury persists as a grave worldwide problem. Some victims succumb fatally to its effects; others who survive after protracted hospital stays with intensive or high care, may yet end up with scars, deformities, contractures, keloids and amputation of digits or limbs. In severe cases, multiple stage reconstructive surgery may be neccessary as palliative rather than curative treatments. .

The morbidity and mortality of burns is due principally to the extent and depth of burns, the age of the victim and multi-system pathology embracing the spectrum from inhalational syndrome to shock, infection and multi-organ failure. The extremes of age - the very young (less than five years) and the older (over fifty) are at greatest risk. A greater depth of understanding of the pathophysiology, revised and more refined intravenous formulae, improved rationale and efficacy of chemotherapeutic agents, gigantic strides in the advance of monitoring equipment and a multi-disciplinary burns personnel have all contributed to the decreasing mortality and morbidity of burns.

The systemic effects of burns involve, generally speaking, the respiratory, cardiovascular and urogenital systems, being influenced by the period of survival preceding death. The local effects of burns too, play a role in the mortality and morbidity. In Great Britain, the Children and Young Persons (Amendment) Mortality Act 1952 has contributed by increasing safety and has hence assisted in reducing the incidence of preventable burns. This applies to restriction of sales of fireworks to children less than sixteen years, safer heating appliances (Heating Appliance and (Fireguards) Act 1952 and plumbing and safer nightwear both for children and adults (Children's Nightwear Regulations CSI 1153, 1964).

The present study analyses the post mortem findings of fatal burn victims within the greater Durban area during 1988.

The following parameters were examined:

1) **Epidemiology:** age, race and sex distribution, etiology, incidence and seasonal variation.

2) Classification of Burns.
3) Assessment of Burns (Extent and Depth).
4) Pathophysiology of Burns.
5) Pathology of Burns.
6) Special types of Burns.
7) Mortality of Burns.
8) Prevention of Burns.

Included in the study is a review of the literature and theory of burn injury and the discussion of the main findings.

The main findings of this study were:

1) The incidence of death due to thermal injury constituted approximately 5% of all medico-legal autopsies.
2) The majority of the fatalities were from the Black race group.
3) Female fatalities outnumber males (by a ratio of 1,05 to 1) especially in suicidal deaths due to burns (2,59:1).
4) Most patients were young and from the economically productive sector of the general population in the 15 to 50 age group.
5) The mean age of victims was 26 years.
6) Most injuries were sustained at home (66,9%) and under accidental circumstances (46,1%).
7) The most common source of injuries was open flame burns (64%). In the 0,1 to 5 years age group, the majority of the victims sustained flame burns.
8) The majority of the burn victims (88%) were hospitalised and died of complications.
9) Pulmonary complications (bronchopneumonia and pulmonay emboli) were the most common cause of death.
10) The importance of a thorough and diligent autopsy is stressed.

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CHAPTER 1

INTRODUCTION

The discovery of fire by Man centuries ago is a significant milestone that altered his diet and lifestyle and accelerated his dormant and rudimentary technology. Coupled to this transformation was the catastrophe of burns. Despite the discovery of many modern alternatives, fire continues to play a paramount role in Man's daily life at home and industry.

The understanding, appreciation and reproduction of fire came slowly to Man, as he learnt very rapidly that fire can also induce injury with accompanying intense pain. Hippocrates, in 430 B.C., showed clinical interest in burns. Documented variations of treatment/medication from "melted old swines' seam mixed with resin" to "warm vinegar or oak bark solution" (1) were suggested by Hippocrates. Aristole displayed a keen interest in the pathogenesis of burns. (1) Greco-Roman treatment on burns, dated seventh century A.D., are echoed by Paulis of Aegina, recommending numerous emollients. The Arabic thoughts on burns were documented in the ninth to tenth centuries, recommending local refrigerants. Fortunately for the patient, this alleviated pain. From the 15th century onwards, the European medical men continued work on burns, with American involvement emerging in the early 1920's.

By the end of the nineteenth century, the pathophysiology of burns had been understood and intravenous fluids administration advocated; by the early 1920's the compensation of fluid and protein losses had been demonstrated by Underhill (1).

The modern era of local burn management became popular in 1942 (1) followed by the "exposure method" after World War II. The exposure method entails cleansing of burn wounds followed by placing the patient in bed exposing the affected area, and allowing it to dry. The recent trend in the multi-disciplinary approach best attained by burns teams, complimented by excellent contributions on treatment, monitoring equipment and persistent fundamental research have all played a significant and contributory role in decreasing mortality and morbidity.

Injuries due to burns remain a global problem. In the USA, more than 2,000,000 people suffer thermal injury annually, of whom 70,000 (3,5%) need to be hospitalised (Schwartz) (2). In Canada, 0,02% of a population of 23,000,000 are admitted annually to hospitals (4).

In common with other types of trauma, thermal injury afflicts children and young adults; most commonly with the prognosis being grave in both children and the elderly.

In the Third World Countries the incidence of burns is higher compared to First World Countries and with the poor medical facilities usually available, the mortality is correspondingly greater. The cost in terms of human life, prolonged morbidity, loss of manpower, human suffering, temporary and permanent disability associated with thermally related injuries, result in a staggering economic and emotional drain on financial and social resources (2).

Numerous factors including the standard of living, level of education, the type of heating and cooking facilities and culture are often the determining factors in the aetiology of burns in various communities. With the thorough understanding of these concepts, prevention can be planned accordingly for the avoidance of the often debillitating and fatal thermal injuries.

In Third World Countries, household fires and heat sources are usually unprotected and close to the ground, thus 80% of burns afflict smaller children, with a higher incidence in the colder months (3).

The ultimate treatment of thermal injuries is prevention. The causation of thermal injuries differs from country to country and often within regions of a state. Complex circumstances and situations precede the burn injury, hence elaborate epidemiological studies should be undertaken to determine the pattern of injuries, with the aim of recommending prevention programs and action that shall serve a protective role. This is applicable particularly in the constantly recurring and predictable burn accident scenarios.

REFERENCES

1. Artz C.P. and Moncrief J.A. (Editors): The Treatment of Burns. Philedelphia W.B. Sanders and Co. 1969 pp 1-21.
2. Curreri P.W. Burns In: Principles of Surgery Ed. Schwartz S.I. 3rd Edition. Mc Graw Hiel 1983.
3. Mickel R. et al: Burns Resuscitation Manual Chapter 9, Pinetown N.B.T.S.
4. Haberal et al: Epidemiology of Burns in Turkey 1987: Burns Volume 13 No. 2. (136-140).
5. Tedeschi C.G., Ackroyd F.W., Eckert W.G. and Tedeschi L.G: Forensic Medicine Volume 1: Mechanical Trauma: 1st Edition Thermal Injury 715-728.

CHAPTER 2

PATIENTS AND METHODS

The material for this study was drawn from one hundred and fifty two burns in 1988. The post mortems were done in the Department of Forensic Medicine, University of Natal, at the South African Police Medico-legal Laboratory between January and December 1988. Various epidemiological characteristics of the cases and medico-legal information were gleaned from the police documentation.

Burn-related deaths are classified as non-natural deaths and medico-legal post mortems are performed on all cases in terms of the Inquest Act (No. 58 of 1959). Patients were referred mainly from the hospitals within the Durban metropolitan area, namely King Edward VIII, Clairwood, Wentworth, Prince Mshiyeni, Addington and private hospitals. Referrals from outside the Durban area were included in this group.

On being informed of the death of a patient by the hospital authority, the South African Police arranges for the collection of the corpse of the deceased in terms of Act No. 51, Section 212 7(d) of the Criminal Procedures Act of 1977. The limitations of this arrangement are:

i) prolonged post mortem interval,
ii) delayed post mortem is performed precluding accurate microbiological and biochemical analyses and the assessment of sepsis in the burn victim.

The following should be borne in mind when performing an autopsy on a burn victim:

i) a short death - autopsy interval obviates the problem of post mortem autolysis which occurs at an accelerated rate in burns.
ii) A short post mortem interval allows meaningful interpretation of bacteriological specimens taken from the burn wound, blood and viscera.
iii) Sampling of tissue for histological examination is recommended to confirm or establish a diagnosis, for example, bronchopneumonia or acute tubular necrosis.

iv) Alcohol, carbon monoxide and other relevant analyses may be submitted if necessary. These may be relevant in the "short survival" cases.

v) Particular attention should be paid to the respiratory, cardio-vascular, gastrointestinal and genito-urinary systems.

A useful adjunct to the post mortem examination was a written summary of the deceased's sojourn in hospital, including clinical course, management and investigation(s) from the attending clinician.

Haemotoxylin and eosin stains were performed on paraffin-embedded tissue sections for histological examination.

CHAPTER 3

EPIDEMIOLOGY

3.1 INTRODUCTION

Heat energy remains an essential requirement for human survival. When used recklessly, it causes crippling, lifelong injury or a fatality (1).

The aetiology and number of burns victims vary from country to country. The standard of living, industrialisation, education, culture and type of heating and cooking facilities are very often the factors determining the causation of burns in different communities (2).

Epidemiological studies of burns are of vital importance and are invaluable for the following reasons: (3)

a) effective prophylaxis or prevention;
b) identification of risk factors;
c) identification of the burn-prone patient;
d) illustration of the pattern of injuries;
e) reduction of the morbidity and mortality;
f) assessment of the efficacy of new modes of therapy;
g) assessment of the provision of adequate medical care:
    i) immediately post burn: 1) the acute phase,
                              2) the ongoing phase,
    ii) rehabilitation and reconstruction:
        1) surgically, this includes prevention of scar development and release of contracture(s), skin grafting and flap creation.
        2) physiotherapy is invaluable in maintaining mobility and strength of the patient and stretching of contractures.

The implications of burns are both social and economic. The impact on the community is substantial due to:

i) loss of working hours;
ii) the exhorbitant cost of hospital treatment and rehabilitation;
iii) personal loss of income;
iv) personal disfigurement and its psychological sequelae;
v) the loss of life - invaluable and irreplaceable (10).

3.2 INCIDENCE OF BURNS

Burns are not regarded as notifiable in South African, hence accurate statistics are sparse and not readily available. Burn incidents and fatalities are limited to individual institutions and at best represent estimates only. Only fatal burn cases are documented as these are regarded as non-natural deaths in terms of the Inquest Act No. 58 of 1959.

In the United Kingdom, statistics are not elaborate either. It is the impression of certain British authors that the overall incidence of burns remains unknown. However, there has been an apparent decrease in burns-related deaths during the past ten years. In contrast, there is a supposed increase in conflagaration with consequent asphyxia, inhalational burns and inhalation of poisonous fumes (6).

In the United States of America, two million persons suffer from burns each year. Most are treated as out-patients, according to several surveys (Mc Dougal and Slade 1978, Schuck 1978, United States Department of Health, Education and Welfare's Accidental Deaths and Injury Statistics 1960, or a rate of 4,3 deaths per 1000,000 population, United States Department of Police Health Services, Division of Vital Statistics 1962). (7) (1).

In Canada, 8000 (0,03%) from a population of approximately twenty three million people (Dorbrkovsky 1984) (1) are admitted to hospital annually for the treatment of burns.

In the United Kingdom, ten thousand burns victims require hospitalisaion for an average of five to six weeks (6).

In Kanpur, India, Gupta and Srivastava state that fatal burns constitute 10,79% of the total medico-legal deaths (13).

In the present study, fatal deaths due to burns-related injuries constituted 5% of the total medico-legal deaths in the greater Durban area.

3.3 CIRCUMSTANCES UNDER WHICH BURNS OCCUR

Most burns are usually accidental but may rarely be homicidal or suicidal in nature.

3.3.1 Accidental Burns

These usually involve children or females at home, in motor vehicle collisions (car catches fire), industrial and splash burns. More that eighty percent of burns are accidental (6)(8).

3.3.2 Suicidal Burns

Self-immolation by burns with highly flammable liquids, usually gasoline or paraffin poured over clothing and set alight, or heated liquids, usually boiling water (13).

3.3.3 Homicidal Burns

i) Highly flammable liquid is poured over the victim and then set alight.

ii) Petrol Bombs/Molotov Cocktail (petrol-filled glass bottle with ignited cloth wick).

iii) The victim may be burned after death has occurred to destroy evidence.

3.3.4 Epileptics

Epileptics contribute a significant group at risk of suffering burn injury. They comprise ten percent of admissions to some burns units. These individuals sustain burns during a convulsion, when they either fall into a fire or start one by overturning a light or heat source. The burns tend to be rather deep (third degree), usually involving the head and hands, as the loss of consciousness accompanying the fit precludes any attempt at escape from the engulfing flames or other heat source (6).

3.3.5 Age

The extremes of age - the very young (less than five years of age) and the older (over sixty years of age) are at greatest risk (1).

In a recent epidemiological study of hospitalised burns in Turkey, fifty seven percent of six hundred and seventeen patients were less than fifteen years of age and forty-three percent were above the age of fifteen years. Further analysis showed that sixty-eight percent were between birth to six years of age, while thirty-two percent ranged from seven to fifteen years of age (1).

In another series in Jordan, patients less than fifteen years made up fifty-five percent of the total number. A total of thirty-three percent of the patients were below the age of five years, with scalds being the cause of eighty-four percent of the injuries. Overall, sixty-nine percent of thermal injuries sustained by patients were caused by scalds (2).

In Australia, the most common age group suffering from injury due to scalds were children ranging from birth to four years, more especially the birth to two year age groups, usually at the child's home with the parents in attendance (4).

Similarly, in Kuwait, children below five years were the main victims, with the incidence maximal at the age of two years (10).

In China, the peak was between one and four years. (Sowemino 1983; Zhi Xiang Zhu et al 1988) (12).

The older age group, although a high risk group, shows a constant incidence in Denmark (11). However, when burns are sustained by this age group, the mortality is usually higher. Other factors that contribute to this high risk are:

i) syncope near a heat source;
ii) smoking in bed;

iii) scalds from leaking hot water bottles;

iv) alcohol (a) increases the chances of accidentally starting a fire;

(b) reduces chances of noticing a fire which has started;

(c) may impair the ability to escape from a fire.

v) atrophied sense of smell and forgetfulness;

vi) faulty heating appliances;

vii) decreased resistance to carbon monixide. (6)

3.3.6 Sex

Various studies indicate a preponderance of burn injury in males. Some series quote a male predominance below the age of fifteen years as being 2:1 (2), whilst others cite a 3:2 ratio (11). The male to female ratio in children is 3:2 while in adults the ratio is 1,7:1 (10).

Between the age of fifteen and sixty four years, males are more prone to the risk of burns than females, probably as males are more active and burn-prone industrially. Of all burns sustained at work, sixty-six percent involve males, while only ten percent involve females. In the over sixty-five year age group, women are three times more prone to burns due to the nature of their attire which catches fire accidentally, in addition to the problem pertaining to the elderly (6).

3.3.7 Seasonal Variation

Although Mickel et al (8) cite a higher incidence in the Third World during the colder climes, Haberal et al report no such seasonal variation in either children or adults in Turkey (1). Bang and Mosbah, in a three year study from 1984 to 1986 in Kuwait, report an absence of seasonal variation (10). However, seasonal variation is reported from China, with burns being minimal in April and at its peak in December-January, the colder months. Flame injuries were the cause in winter and scalds predominated in summer, as minimal clothing worn by children in summer offered little or no protection against spilled hot liquids (12).

3.3.8 Contributory Factors in the Occurrence of Burns

Factors contributing to the development of burn injuries were noted in sixty percent of the patients in one series (Byron et al 1984) (9). These pre-existing or co-existent factors were considered to decrease the victim's ability to recognise and/or react to the potential or true danger presented by a heat source. The most common contributing factor with burn injury, in the series cited above, was the consumption of alcohol (26% of cases). The presence of contributory factors indicated a greater severity of burn injury compared to victims without contributory factors.

Contributing Factors in Patients with Open Flame/Burn Injuries (9)

	%
Young age (less than six years)	13,5
Old age	35,6
Alcohol	43,3
Physical Disability	25,0
Mental Disability	10,6
Drugs	2,9
Assault	2,9
Self Infliction	5,6
Other	8,7
	100%

3.3.9 FINDINGS IN THE PRESENT SERIES

The epidemiological findings in this series were as follows:

3.3.9.1 Racial Distribution

Blacks constituted the majority of the victims (87%); Asians accounted for 6,5%; Whites 5,8% and Coloureds 0,6% of burn fatalities. (Refer to FIGURE 3.1).

The higher incidence of burns in Blacks may be explained on the basis of one or more of the following:

i) lower socio-econimic standing;
ii) the use of unguarded, floor level cooking and heating methods;
iii) overcrowding;
iv) the lack of piped hot water;
v) use of pressure stoves;
vi) the falling of candles onto beds or other flammables;
vii) the high level of unrest in the townships, with petrol-bombing of homes with occupants within, and "necklacing";
viii) the lack of safety measures at home.

RACIAL DISTRIBUTION 1988

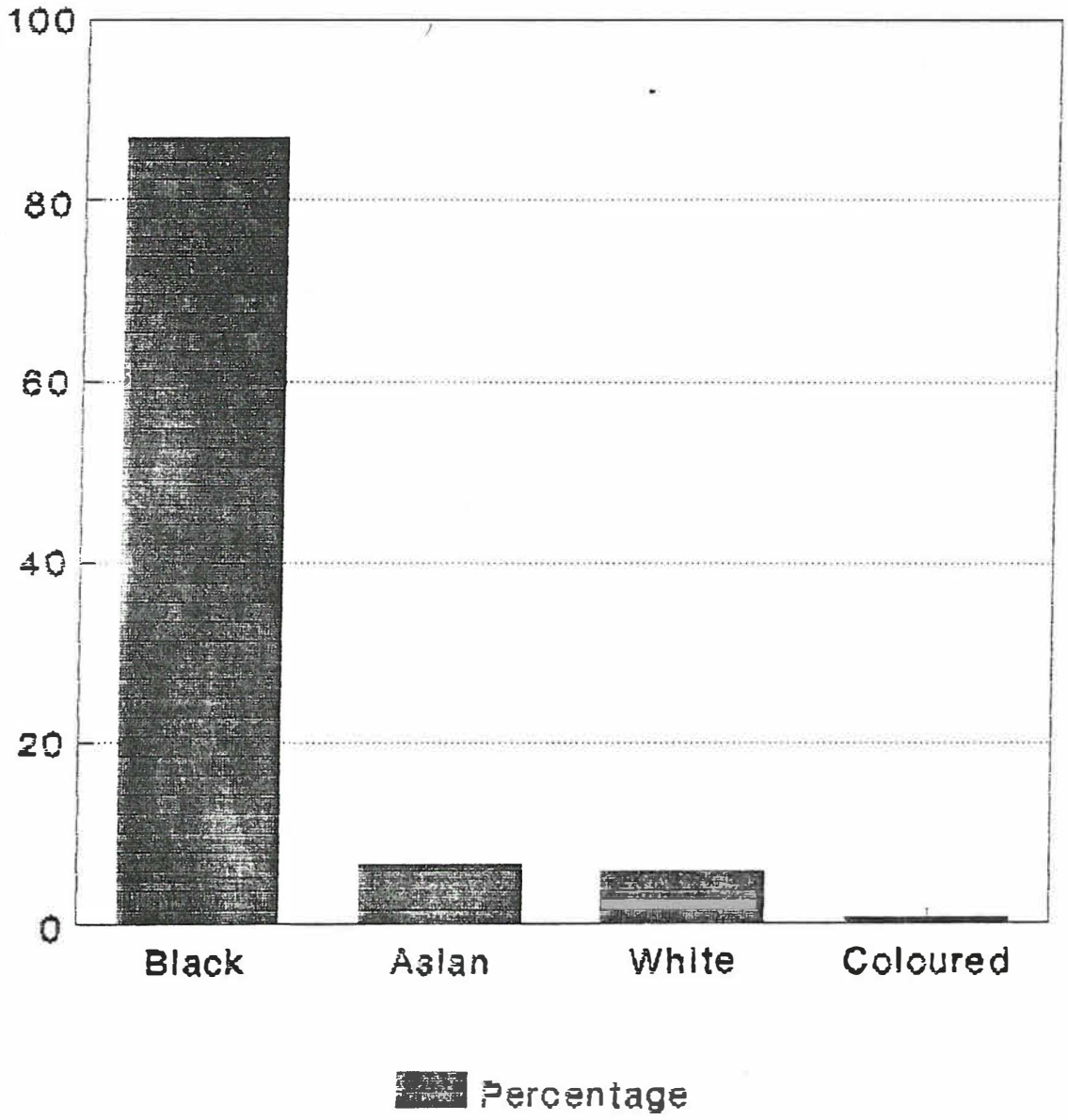


FIGURE 3.1

3.3.9.2 Population Distribution in Durban

The population census held in 1985 showed the racial distribution in Durban as follows: (TABLE 3.1)

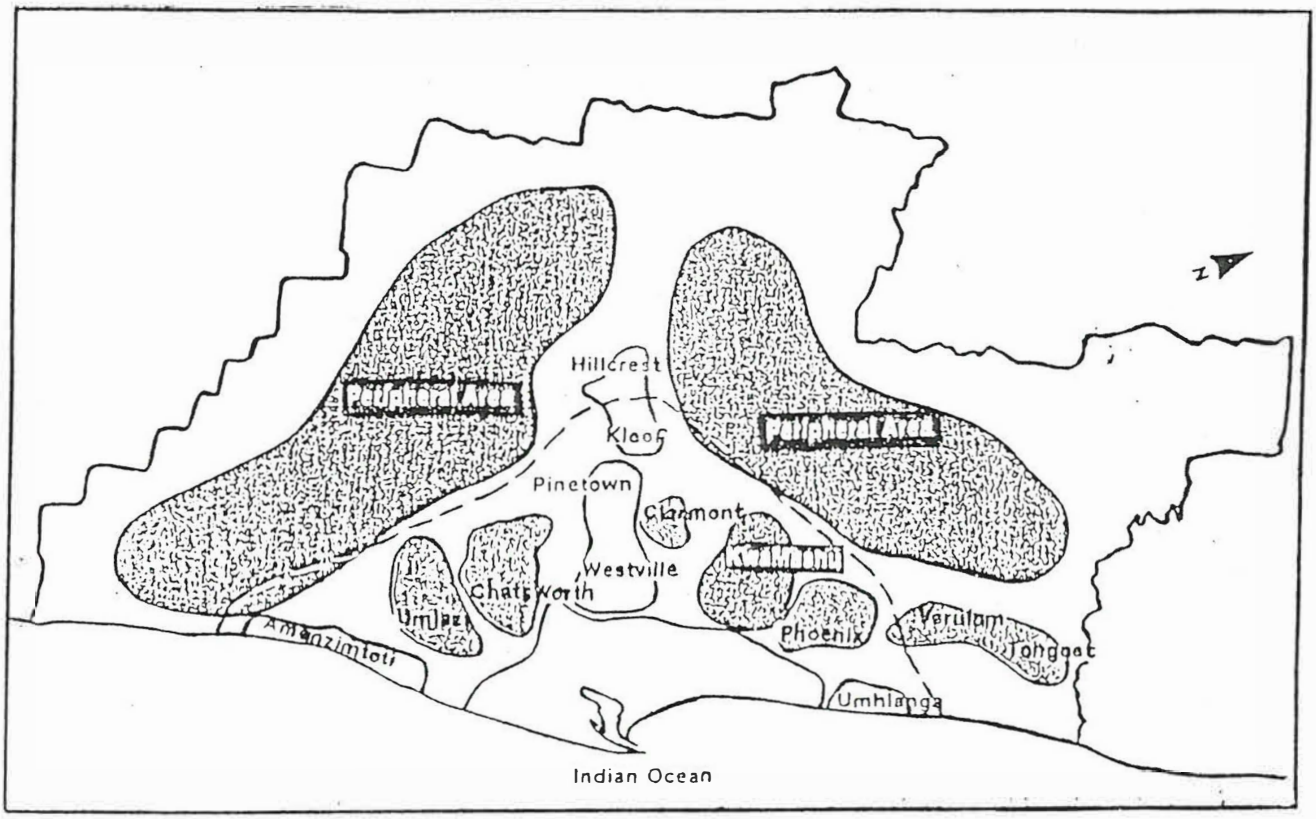
TABLE 3.1 - RACIAL DISTRIBUTION ACCORDING TO POPULATION CENSUS 1985

AREA	ASIANS	WHITES	COLOUREDS	BLACKS	TOTAL
DURBAN CITY	101004	173145	39188	32878	346215
DURBAN METRO-POLITAN AREA	372000	17368	54465	33973	634301
PERCENTAGE OF METROPOLITAN POPULATION	58,7	27,4	8,6	5,46	

(Source: Central Statistical Services, Population Census 1985)

The proportion of burn-related fatalities derived from the different race groups is not proportional to the distribution table above (TABLE 3.1). The number of White burn victims is a true reflection of burn fatalities in that group. Burn-related medico-legal autopsies involving Blacks, Asians and Coloureds are performed at peripheral mortuaries situated in the magisterial districts of Chatsworth and Verulam. The Medico-legal Laboratory in Durban services the metropolitan Durban area, including the Kwa Zulu enclave of Umlazi located south of Durban and the so-called "peripheral areas". (Refer FIGURE 3.2). Thus, a disproportionately higher number of Blacks are represented.

FIGURE 3.2 - METROPOLITAN DURBAN AND PERIPHERAL AREAS



3.3.9.3 Sex Distribution

Males comprised 48,7% and females 51,3% of burns fatalities. The female to male prooortion is 1,05 : 1,0. (Refer to FIGURE 3.3).

The marginally higher female preponderance may be ascribed to the higher rate of suicidal burns in females (72,2%) and also because women spend more time at home, where the incidence of accidental burns is highest. (Refer FIGURE 3.10).

SEX DISTRIBUTION
1988

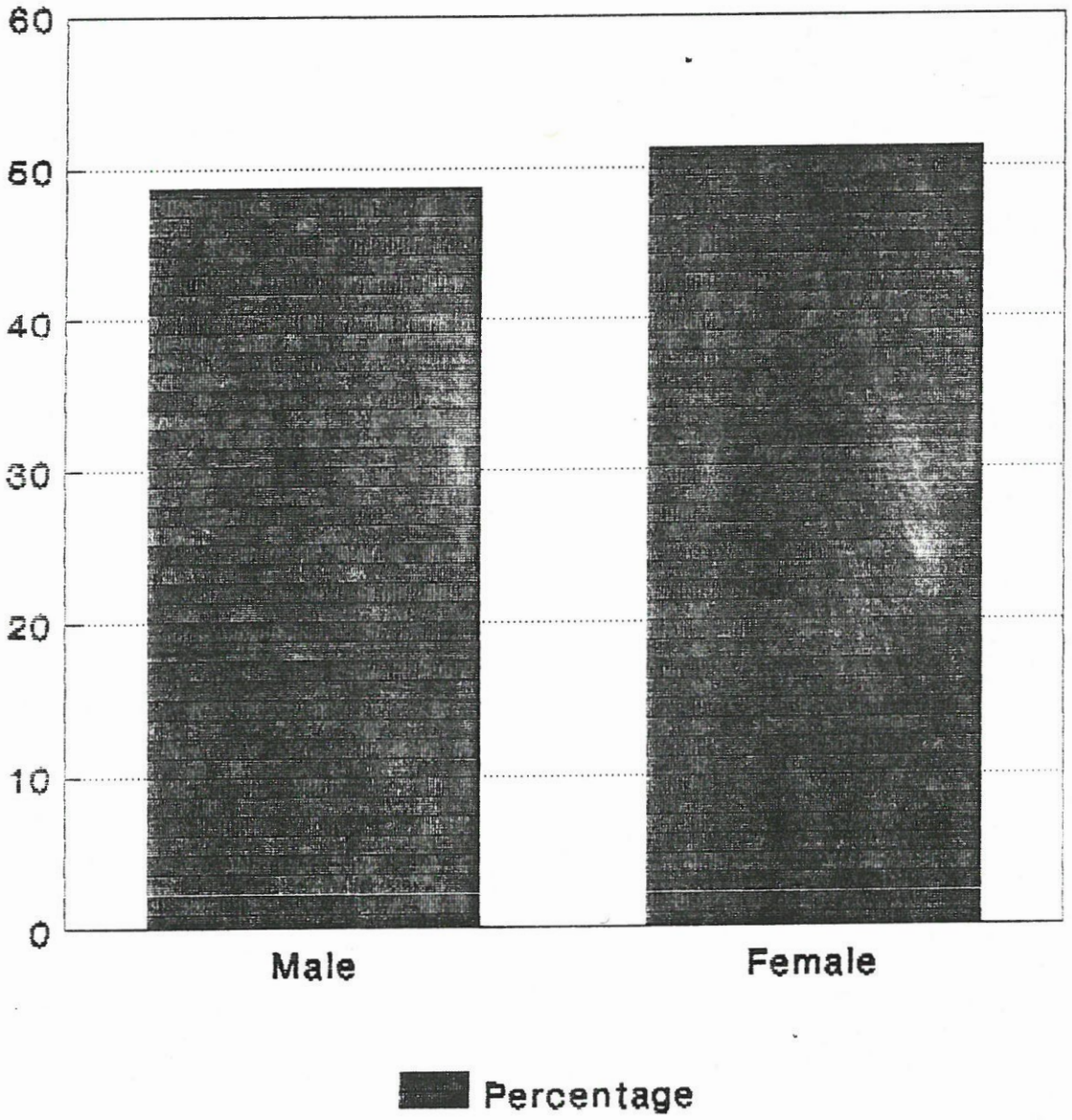


FIGURE 3.3

3.3.9.4 Age Distribution

The age distribution of fatal burns is depicted graphically in FIGURE 3.4. A major peak is present in the 16-50 year age group and a second lesser peak in the 1-5 year age group. The ages ranged from two months to eighty-seven years, with a mean age of twenty-six years.

The economically active young adult and children constitute the majority in fatal burns.

Unlike other series elsewhere, (1)(10)(16), the maximum incidence of injury in this series was the 16-50 year age group (young adults). The low incidence (11,7%) in the high risk, burn-prone (greater than fifty years) and the high incidence in the 16-50 year age group is peculiar to this series.

In the 0,1 - 5 year age group, forty-two children died; 51,3% died due to flame burns; 30,7% died due to scalds (Refer to FIGURE 3.5). This is contrary to the findings of other authors (1)(6)(10)(16).

The race and sex distribution in the 0,1 - 5 year age group is as follows:

AGE DISTRIBUTION
1988

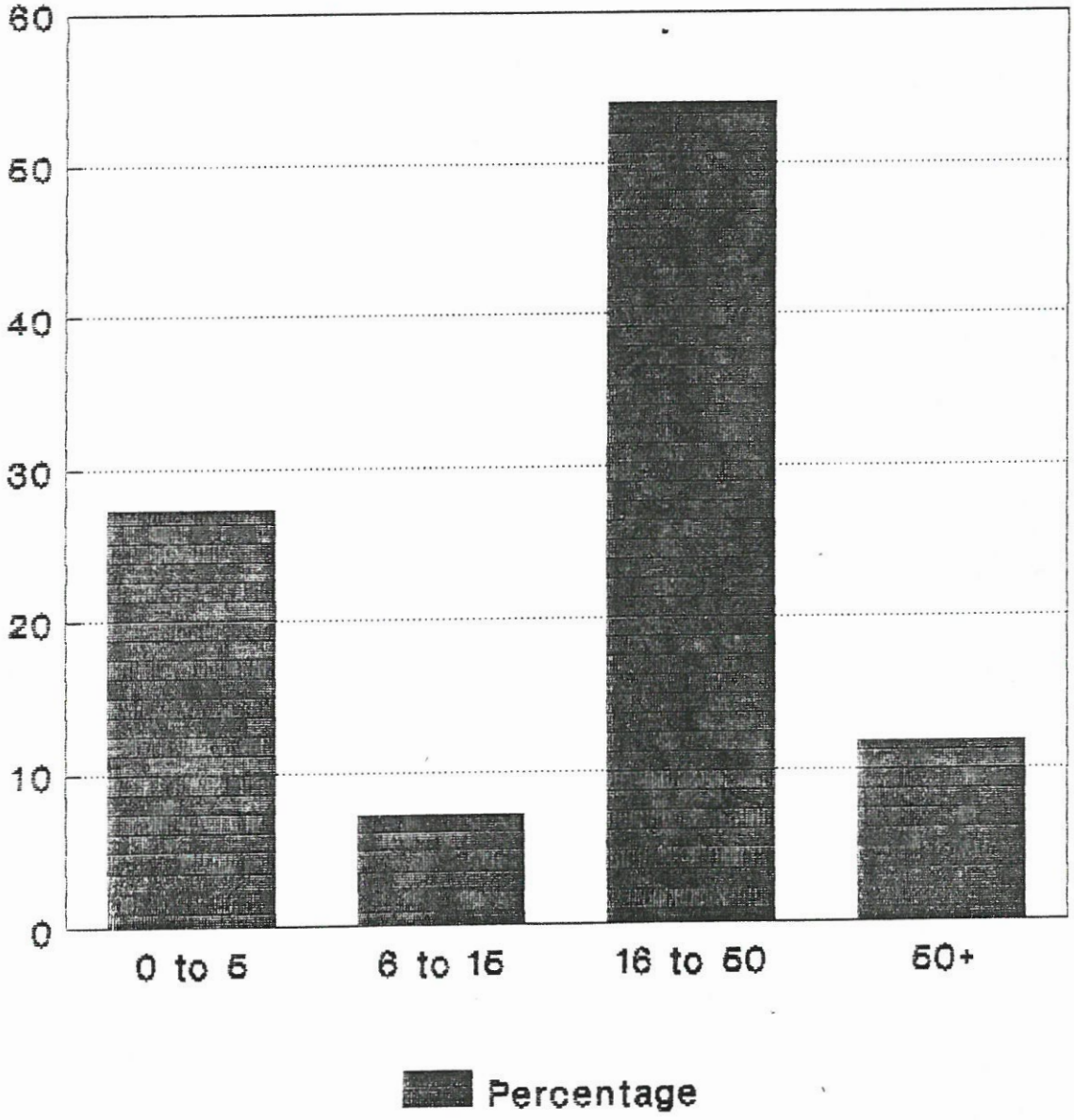


FIGURE 3.4

SOURCE OF BURN
AGE GROUP < 5

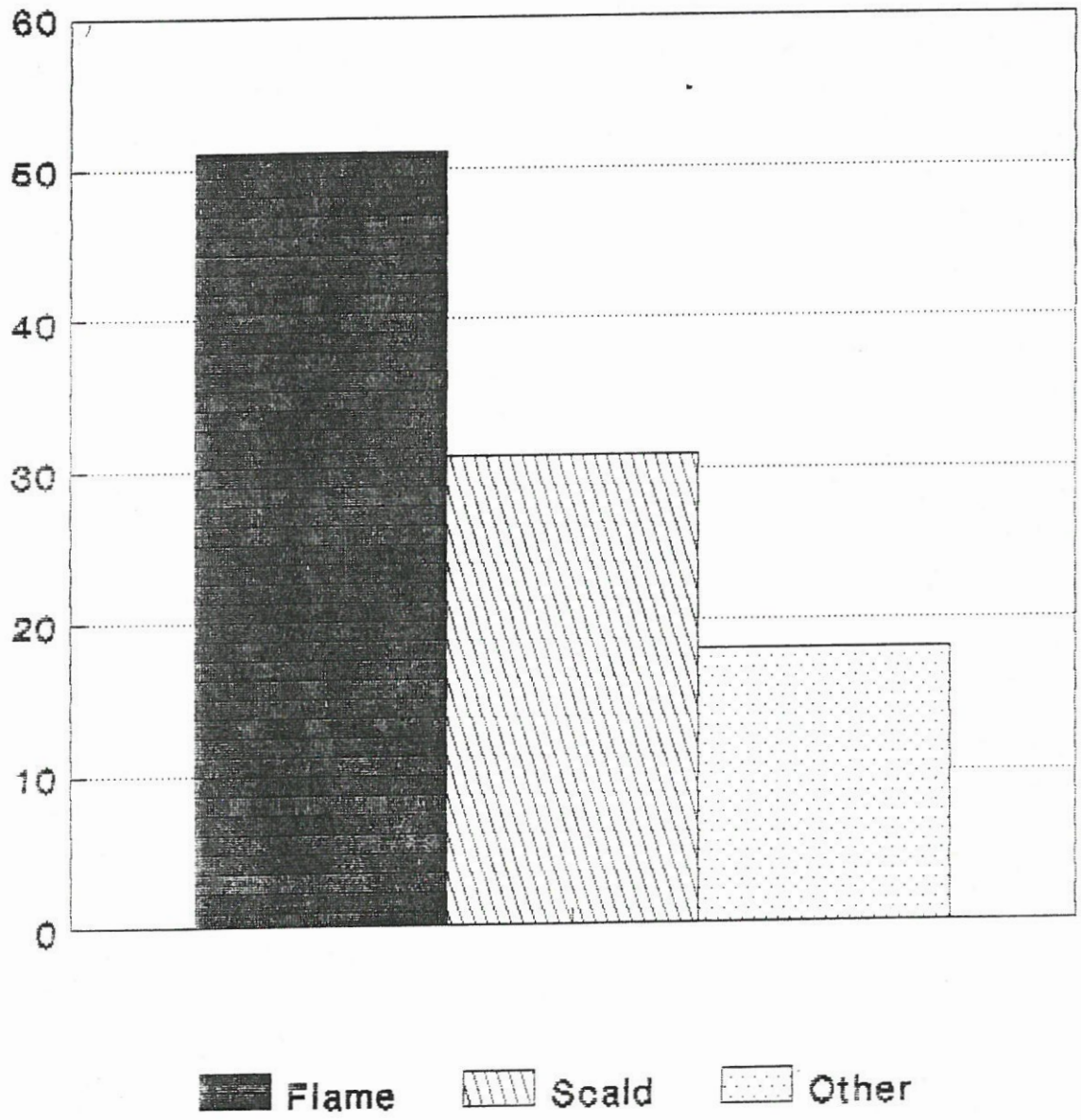


FIGURE 3.5

TABLE 3.2 - RACIAL DISTRIBUTION FOR AGE GROUP 0,1 - 5 YEARS

RACE	BLACKS	ASIANS	WHITES	COLOUREDS
PERCENTAGE	92,3	5,2	2,6	0
RATIO	35,5	1,96	1,0	0

FIGURE 3.6 REFERS

TABLE 3.3 - SEX DISTRIBUTION FOR AGE GROUP 0,1 - 5 YEARS

SEX	FEMALE	MALE
PERCENTAGE	53	46
RATIO	1.1	1

REFER TO FIGURE 3.7

RACIAL DISTRIBUTION FOR AGE GROUP < 5

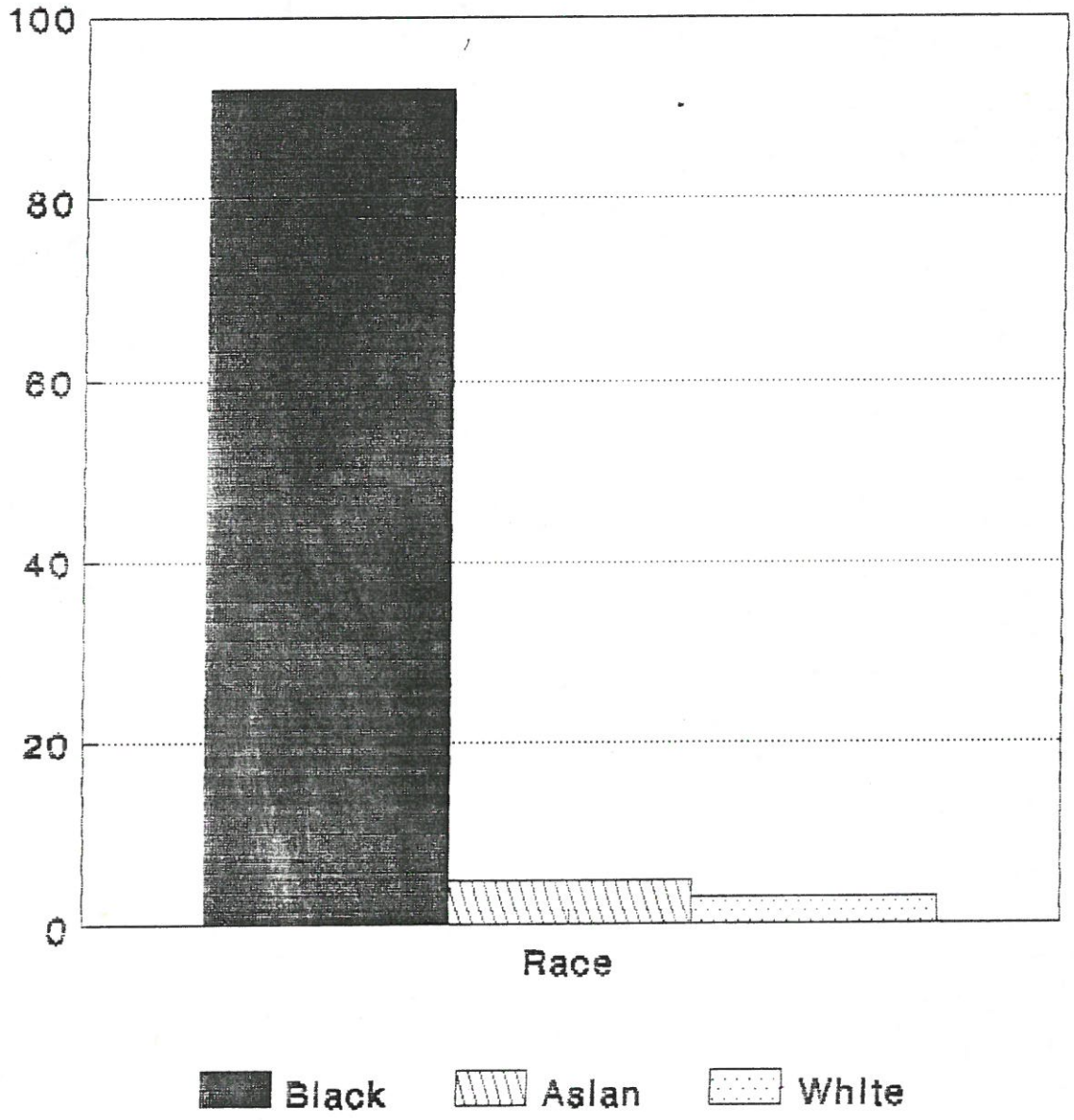


FIGURE 3.6

SEX DISTRIBUTION
FOR AGE GROUP < 5

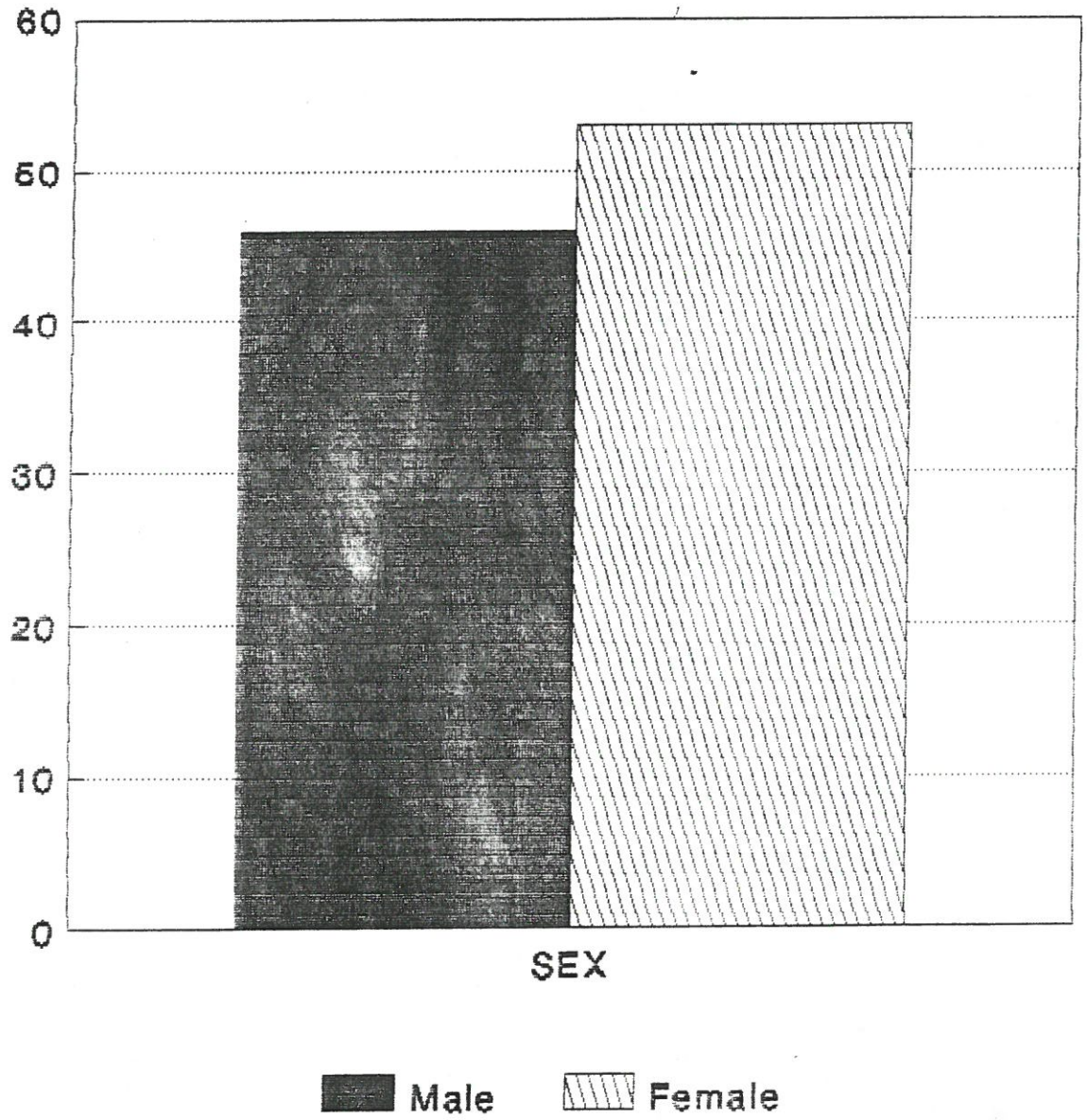


FIGURE 3.7

3.3.9.5 Place of Injury

Figure 3.8 depicts graphically the site/place of injury. As expected and documented, (1)(10)(16), most burn fatalities occur at home.

TABLE 3.4 - PLACE OF INJURY IN FATAL BURNS

PLACE OF INJURY	HOME	WORK	CAR	OTHER
FREQUENCY (%)	66,9	3,9	3,2	3,9

PLACE OF INJURY
1988

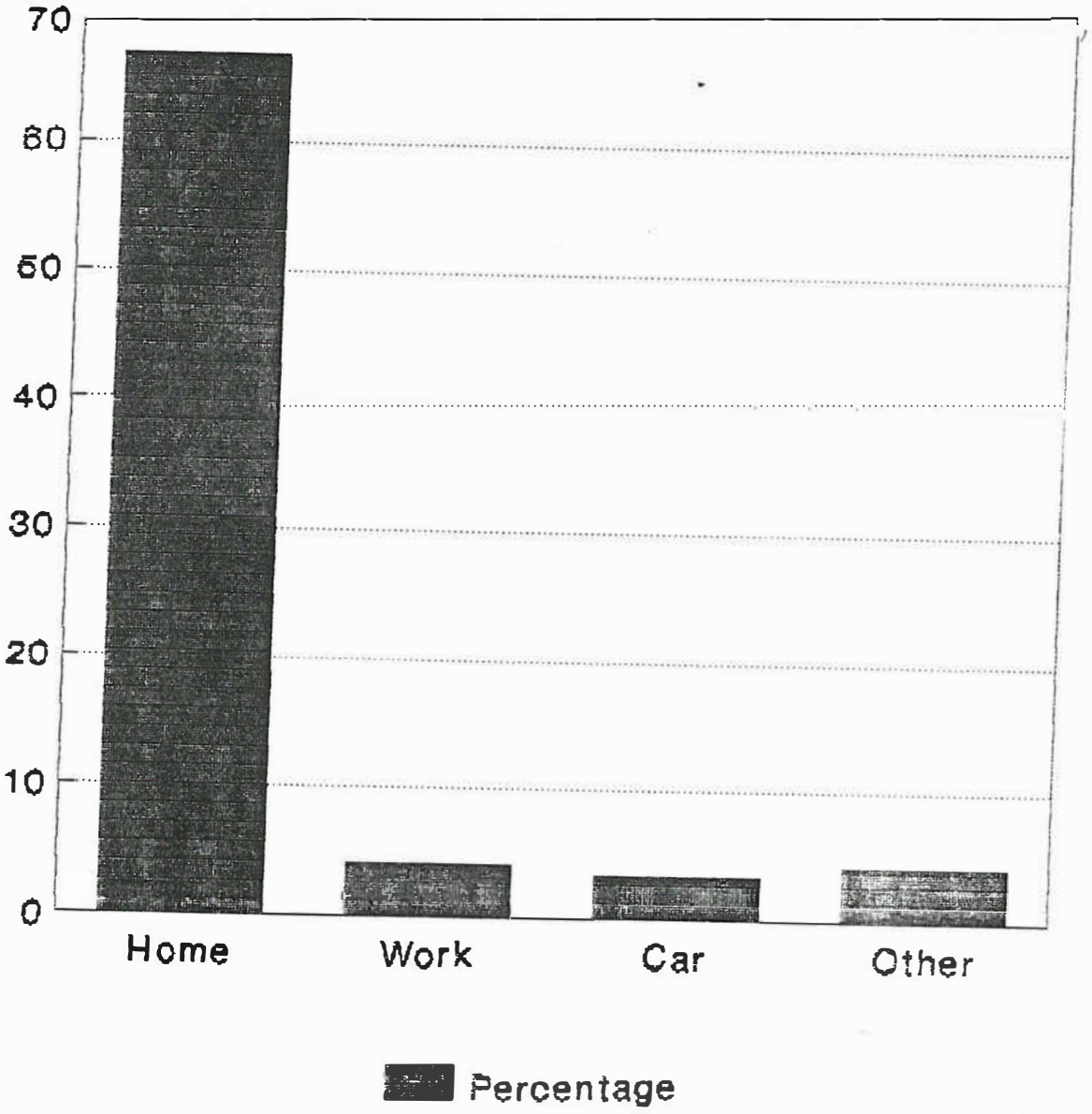


FIGURE 3.8

3.3.9.6 Circumstance of Injury

In this series, most burns injuries were sustained accidentally (46,1%); homicidal and suicidal burns accounted for 11,7% each. Refer to FIGURE 3.9.

The number of victims that committed suicide totalled 18 or 11,6% of the sample. The female to male ratio of burn fatalities was 72,2% : 27,8% or 2,6 : 1. (Refer to FIGURE 3.10).

TABLE 3.5 - SUICIDAL BURNS

RACE	BLACKS	ASIANS	WHITES
PERCENTAGE	83,3	11,1	5,5
RATIO	15	2	1

REFER TO FIGURE 5.11 FOR GRAPHICAL ANALYSIS

i) Age Range: 15-65 years;

ii) Circumstance: Paraffin - 66,7%
Petrol - 16,6%

iii) Mean age: 30,8.

The high frequency of suicide by fire may be explained by the ready availability of paraffin in the home and ignorance of other methods of suicide. The profile of a burn-related suicide in this series is a young Black female, aged ±30, using paraffin for self-conflagaration. The youngest victim of suicide by burning was fifteen years old (female) and the oldest was sixty-five years of age (male).The maximum incidence of burns was in the 16-50 year age group.

CIRCUMSTANCE OF INJURY

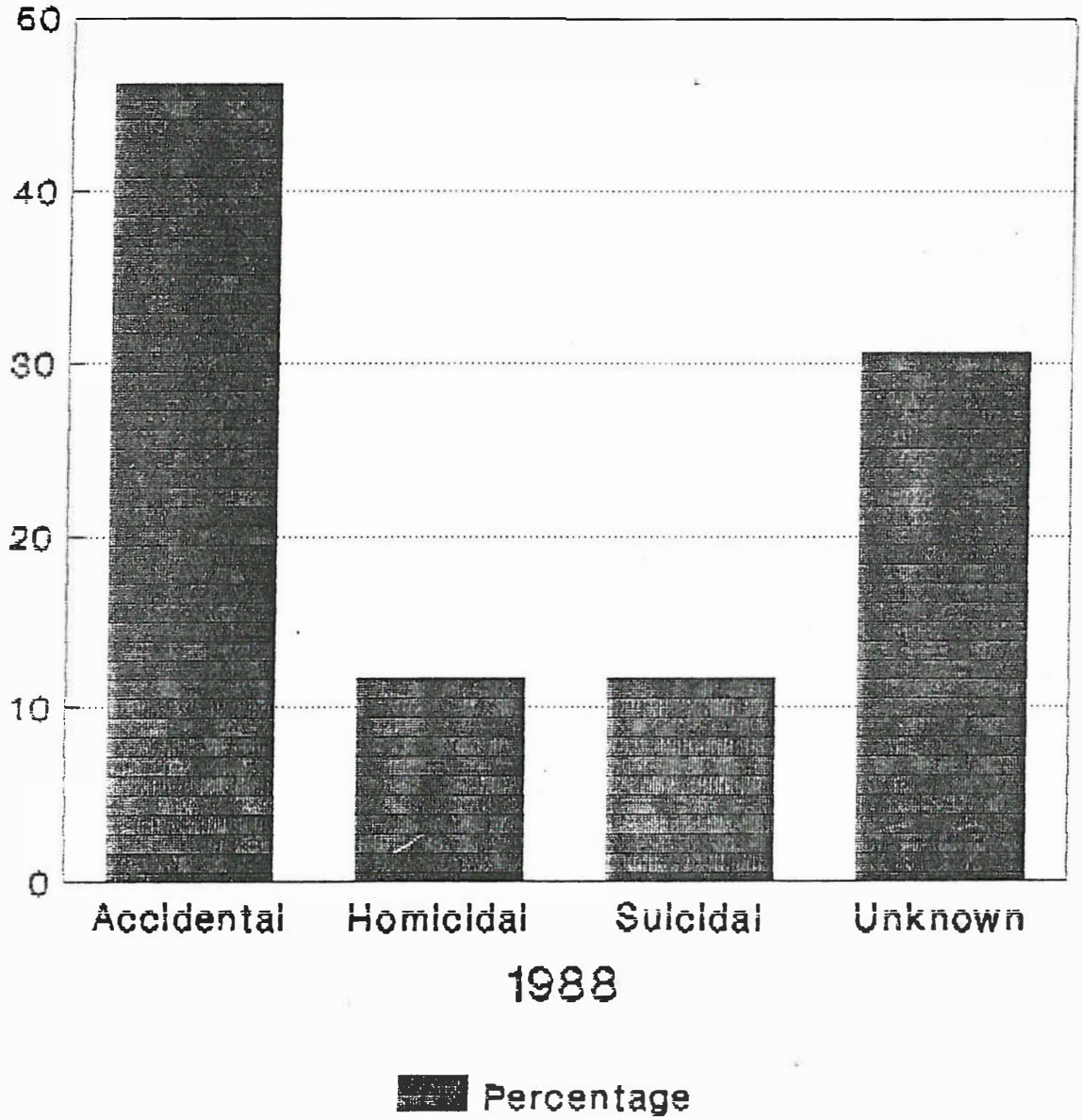


FIGURE 3.9

SUICIDAL BURNS
1988

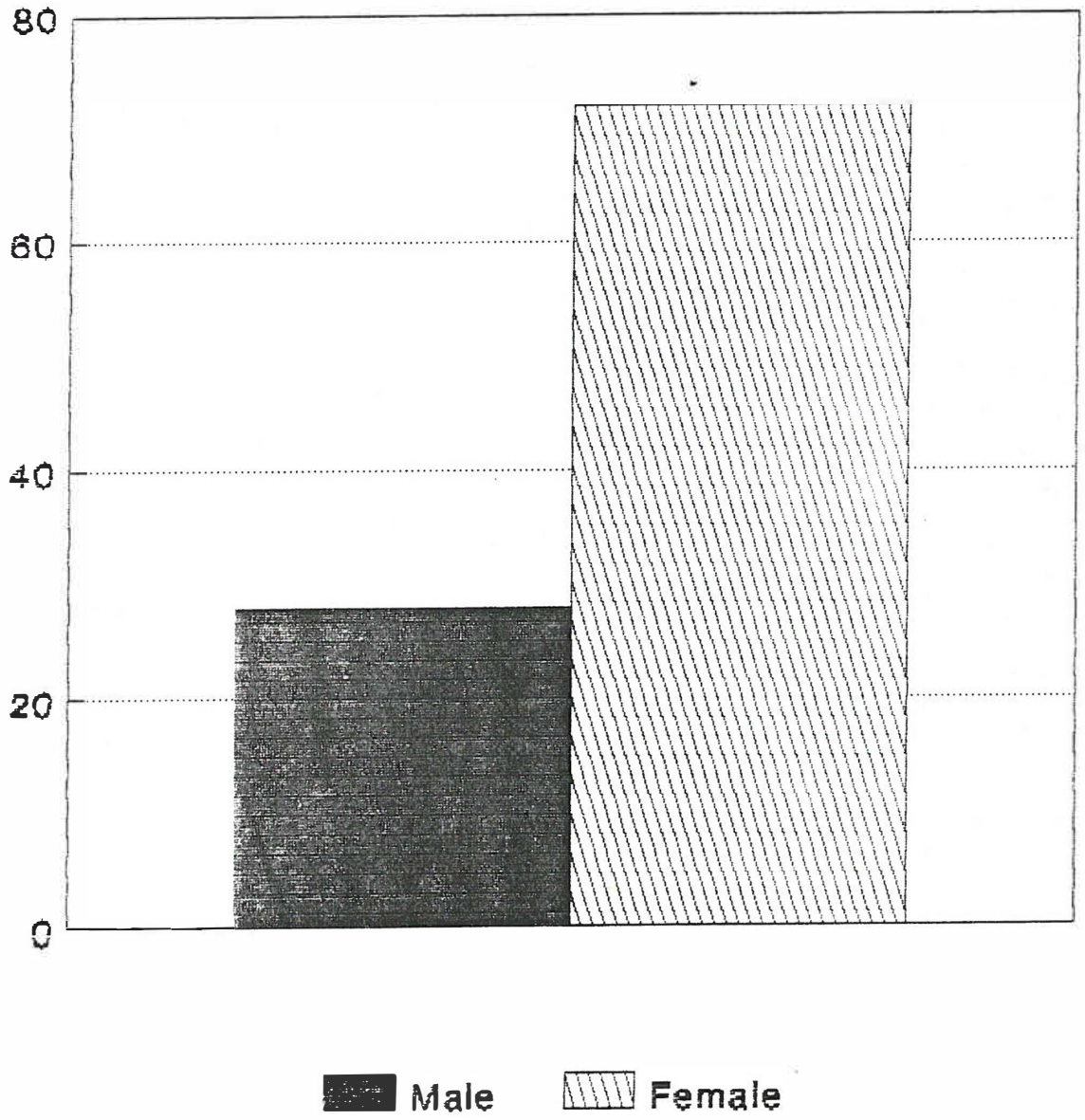


FIGURE 3.10

SUICIDAL BURNS
1988

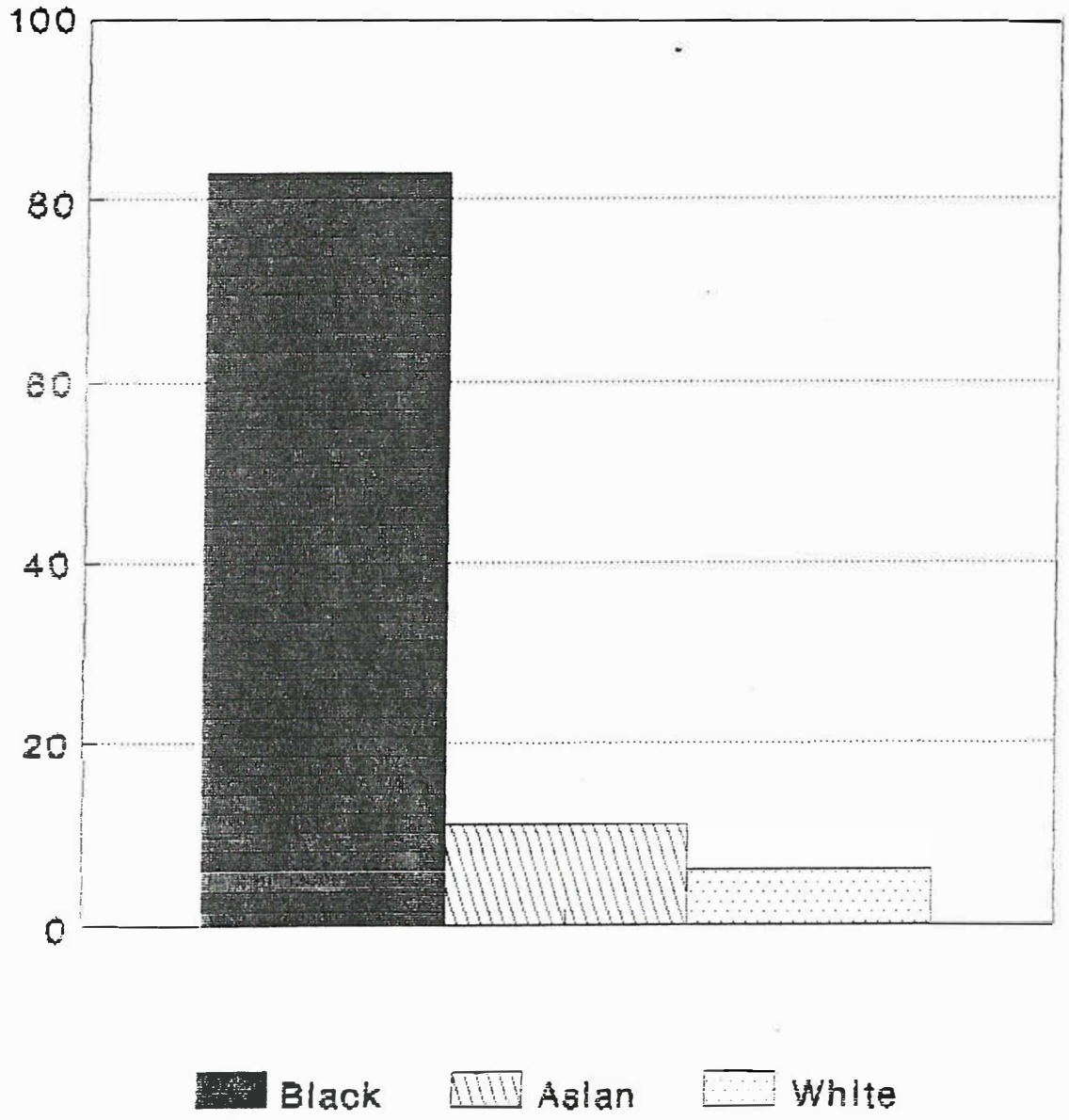


FIGURE 3.11

3.3.9.7 Source of Injury

FIGURE 3.12 illustrates graphically that flame burns were responsible for 64% of all burn fatalities, scalds for 10,4%, electrical injuries for 3,9% and chemical injuries for 0,6%.

TABLE 3.6 - SOURCE AND FREQUENCY OF BURN INJURIES

SOURCE OF INJURY	FREQUENCY (%)
FLAME	64
SCALD	10,4
ELECTRICITY	3,9
CHEMICAL	0,6
OTHER	20

SOURCE OF INJURY
1988

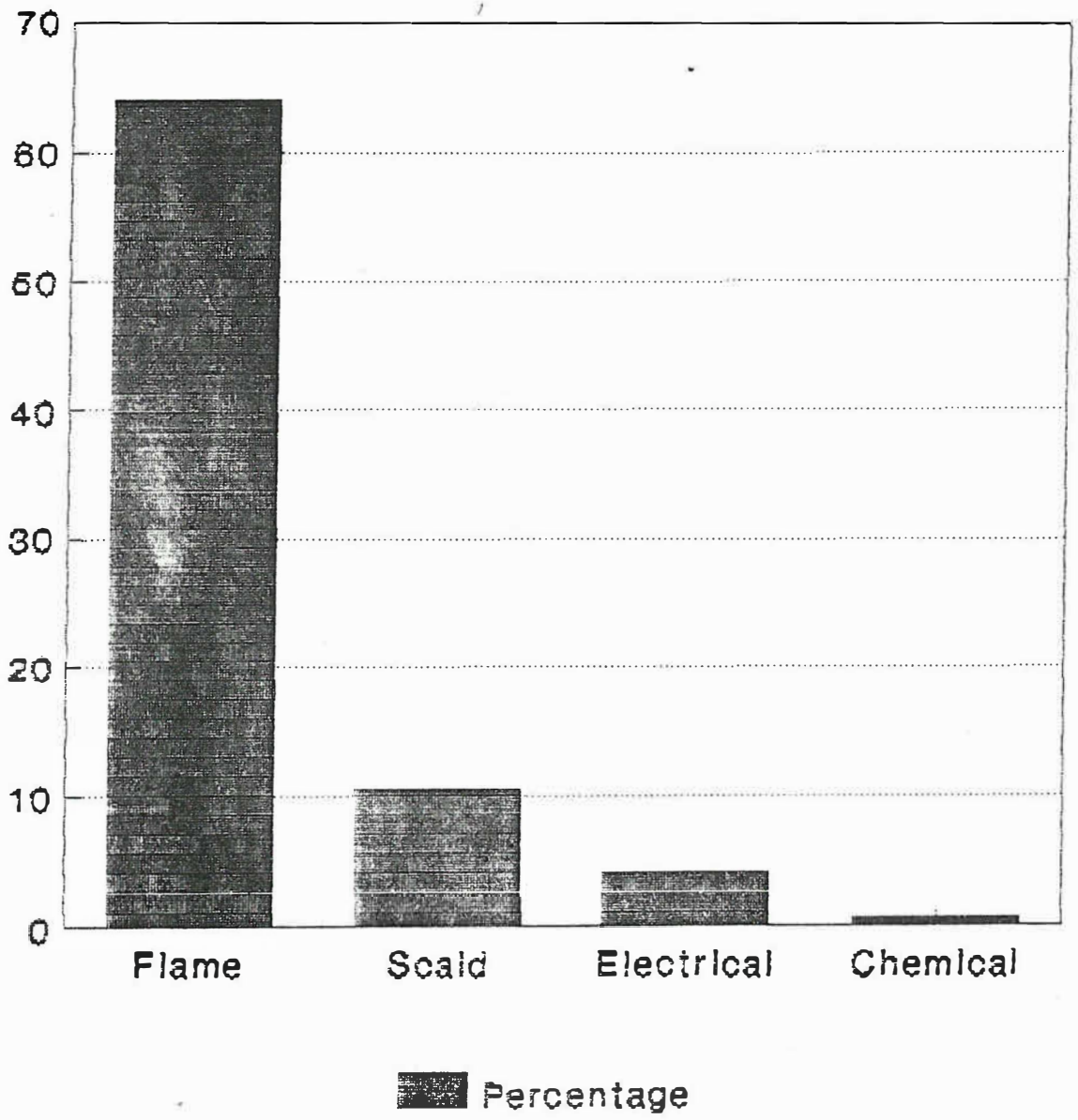


FIGURE 3.12

3.3.9.8 Hospital Admission

The vast majority of fatalities (88%) in this study of 151 victims survived the burn episode, to be admitted to hospital and died due to the complications of burns. (Table 3.7 refers).

TABLE 3.7 - COMPLICATIONS OF BURNS

PERCENTAGE	COMPLICATION
39,0	BRONCHOPNEUMONIA
17	ACUTE TUBULAR NECROSIS
13	INHALATIONAL INJURY
6	SHOCK

The minority (12%) of victims studied died at the burns scene and prior to admission. These cases represent severe burn injuries and death was caused by carbon monoxide poisoning, inhalational injury, irreversible hypovolaemic shock and charring.

FIGURE 3.13 is a graphical presentation of hospital admission.

HOSPITAL ADMISSION 1988

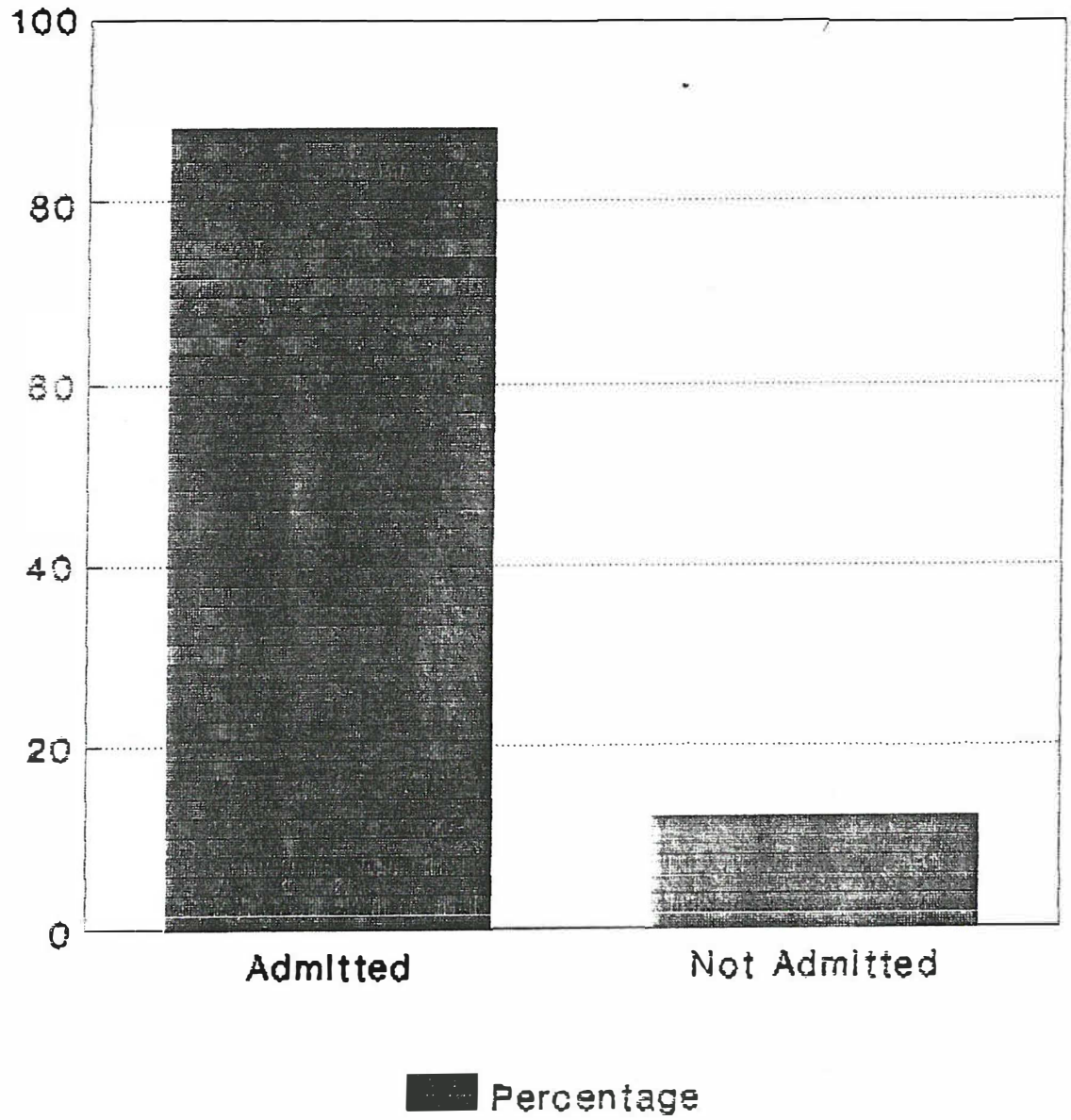


FIGURE 3.13

3.3.9.9. Extent of Injury

The extent of body surface area burns in fatal cases is graphically illustrated in FIGURE 3.14

TABLE 3.8 - EXTENT OF INJURY IN BURN VICTIMS

% BURNS	% OF CASES
LESS THAN 10	13,6
11-30	26,0
31-50	20,8
51-100	51,6

EXTENT OF INJURY
1988

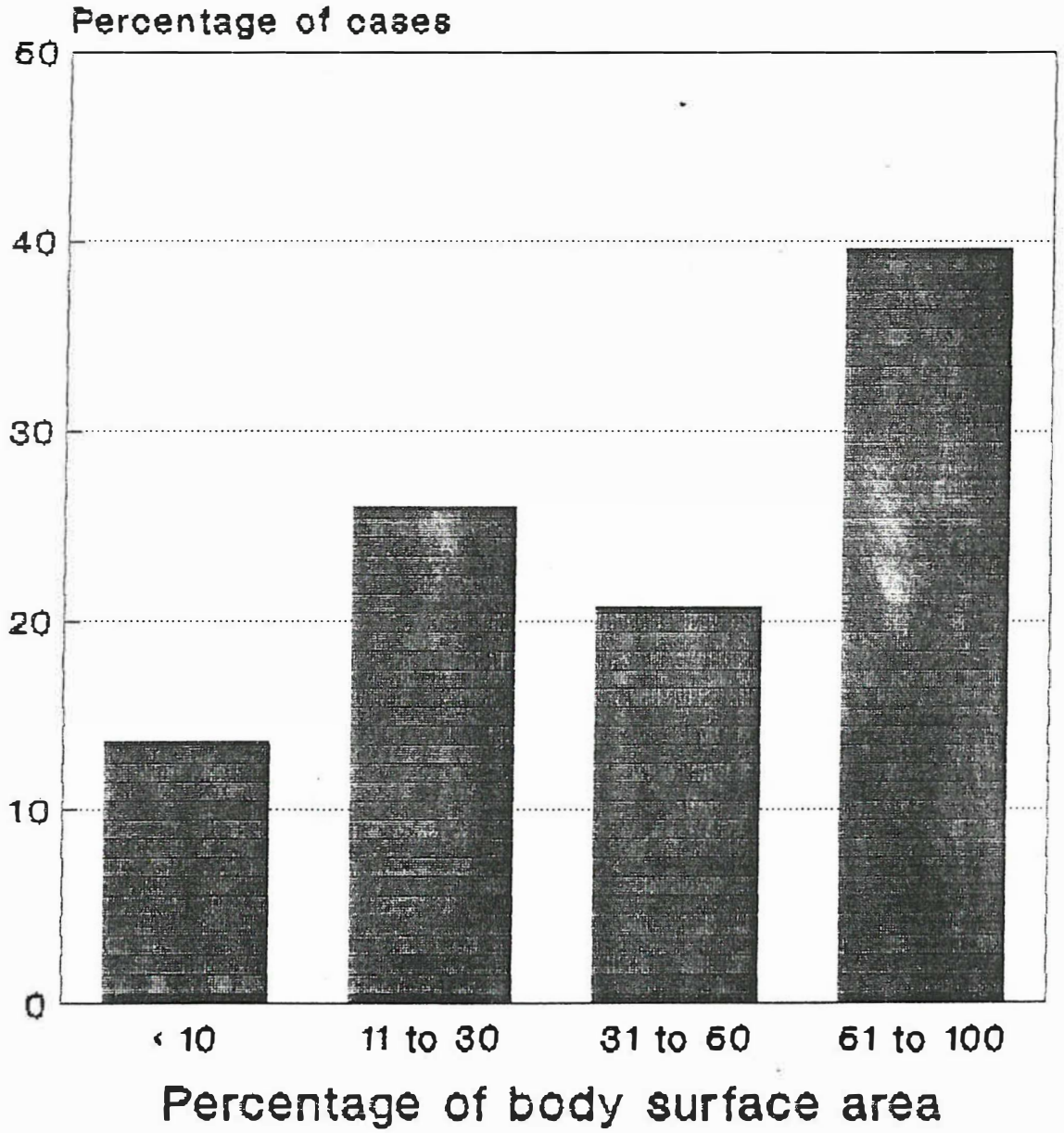


FIGURE 3.14

3.3.9.10 Depth of Burn Injury

TABLE 3.9 tabulates and FIGURE 3.15 graphically illustrates the degree of burn injuries.

TABLE 3.9 - DEGREE OF BURN INJURIES

Degree of Burn	Percentage of Cases
Second Degree Burns	25
Second and Third Degree Burns	45
Third Degree Burns	42
Fourth Degree Burns	65

TYPE OF INJURY
1988

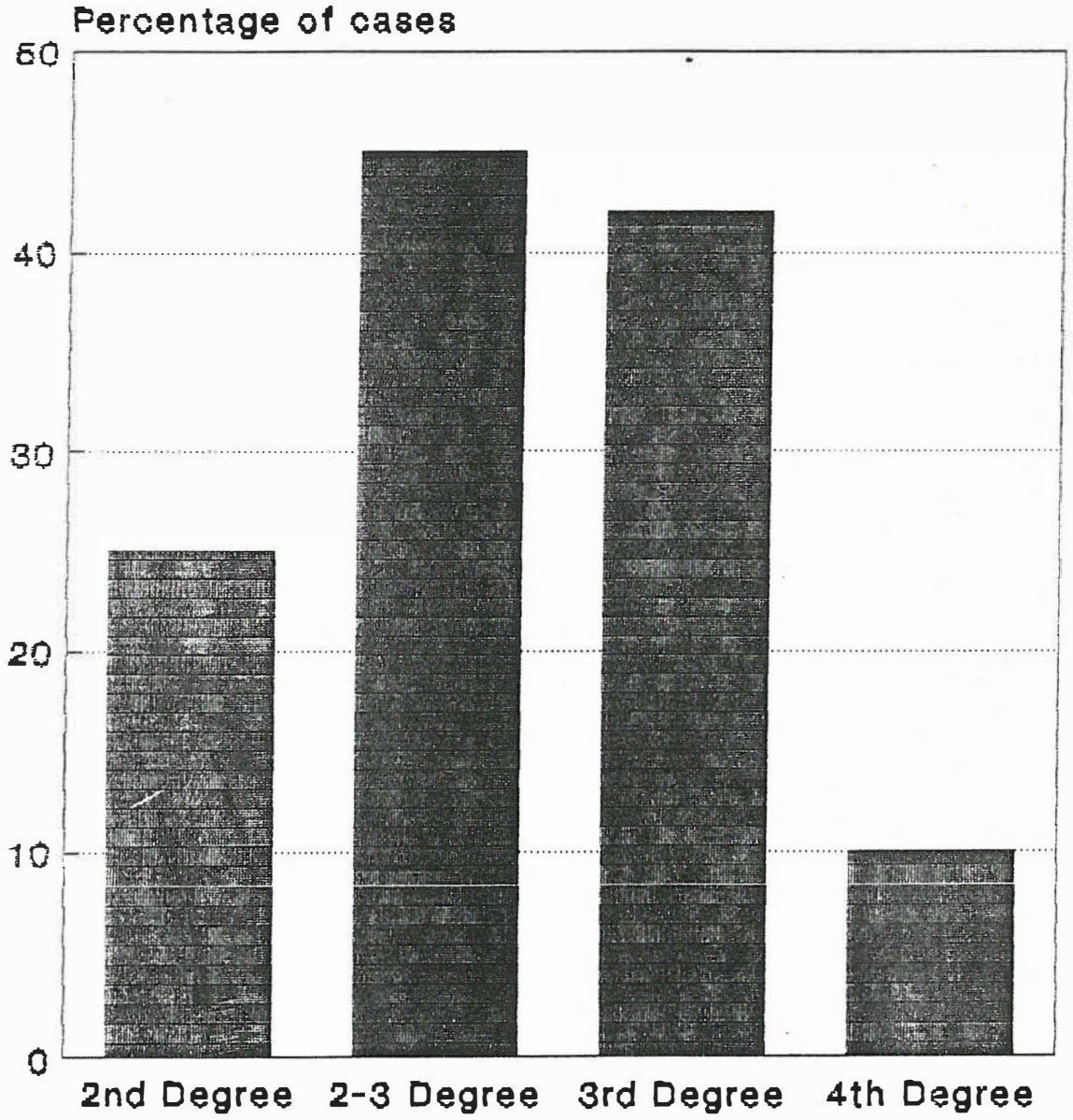


FIGURE 3.15

3.3.9.11 Anatomical Areas of Body Affected

FIGURE 3.16 graphically represents the anatomical areas of the body commonly affected in burn injuries. (TABLE 3.10 refers).

TABLE 3.10 - ANATOMICAL AREAS OF THE BODY COMMONLY AFFECTED IN BURN INJURIES

AREA OF BODY	PERCENTAGE OF VICTIMS AFFECTED
UPPER LIMBS	86,4
LOWER LIMBS	80,5
HEAD AND NECK	72,0
TRUNK	70,0
ABDOMEN	68,0
BACK	61,0

AREAS OF BURNS
1988

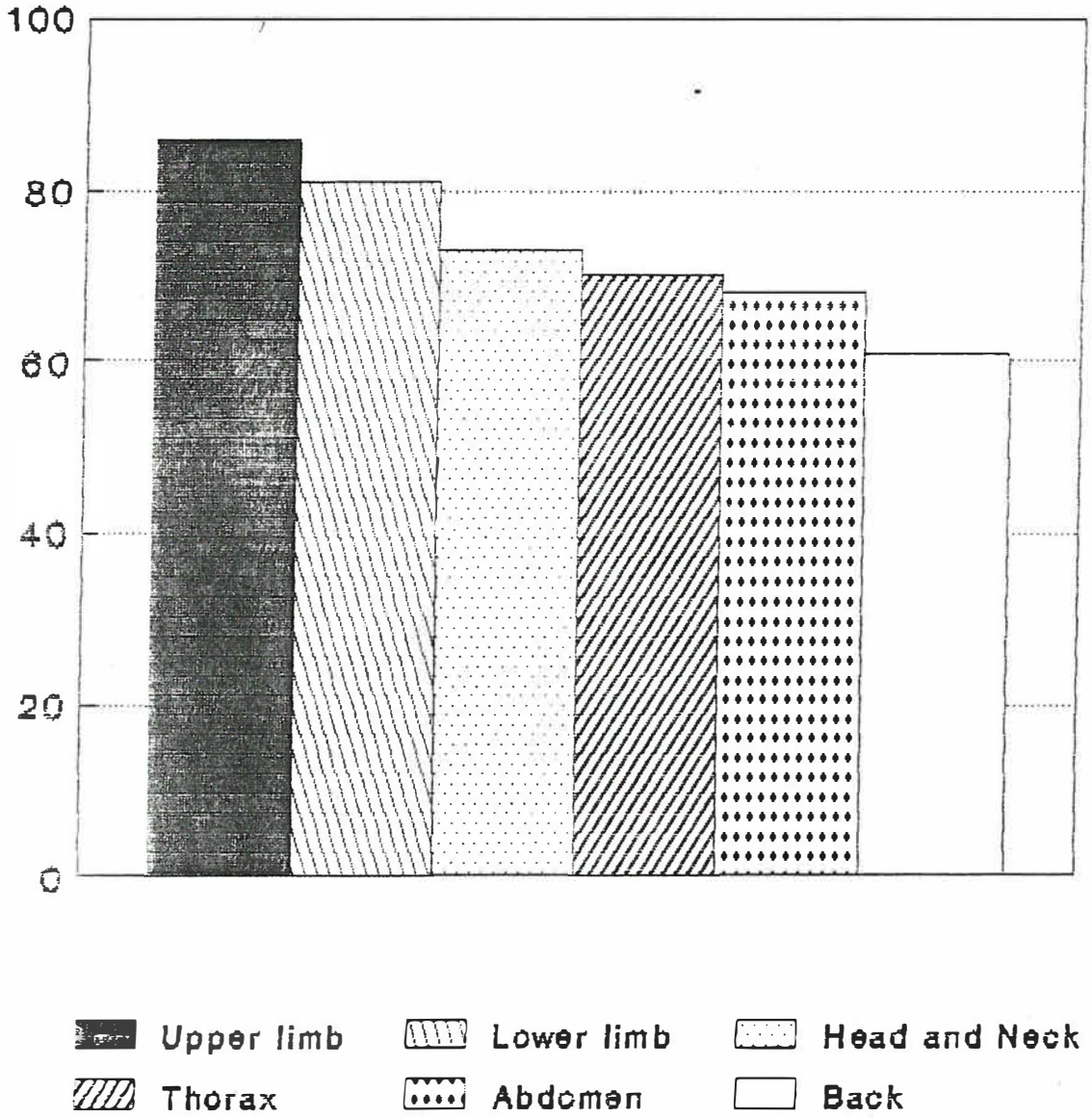


FIGURE 3.16

3.3.9.12 Seasonal Variation

In the present series, no significant statistical seasonal variation in the incidence of burns was evident. There was a slightly higher incidence during the colder months and a lower incidence in the warmer months. (FIGURE 3.17 refers).

SEASONAL VARIATION
1988

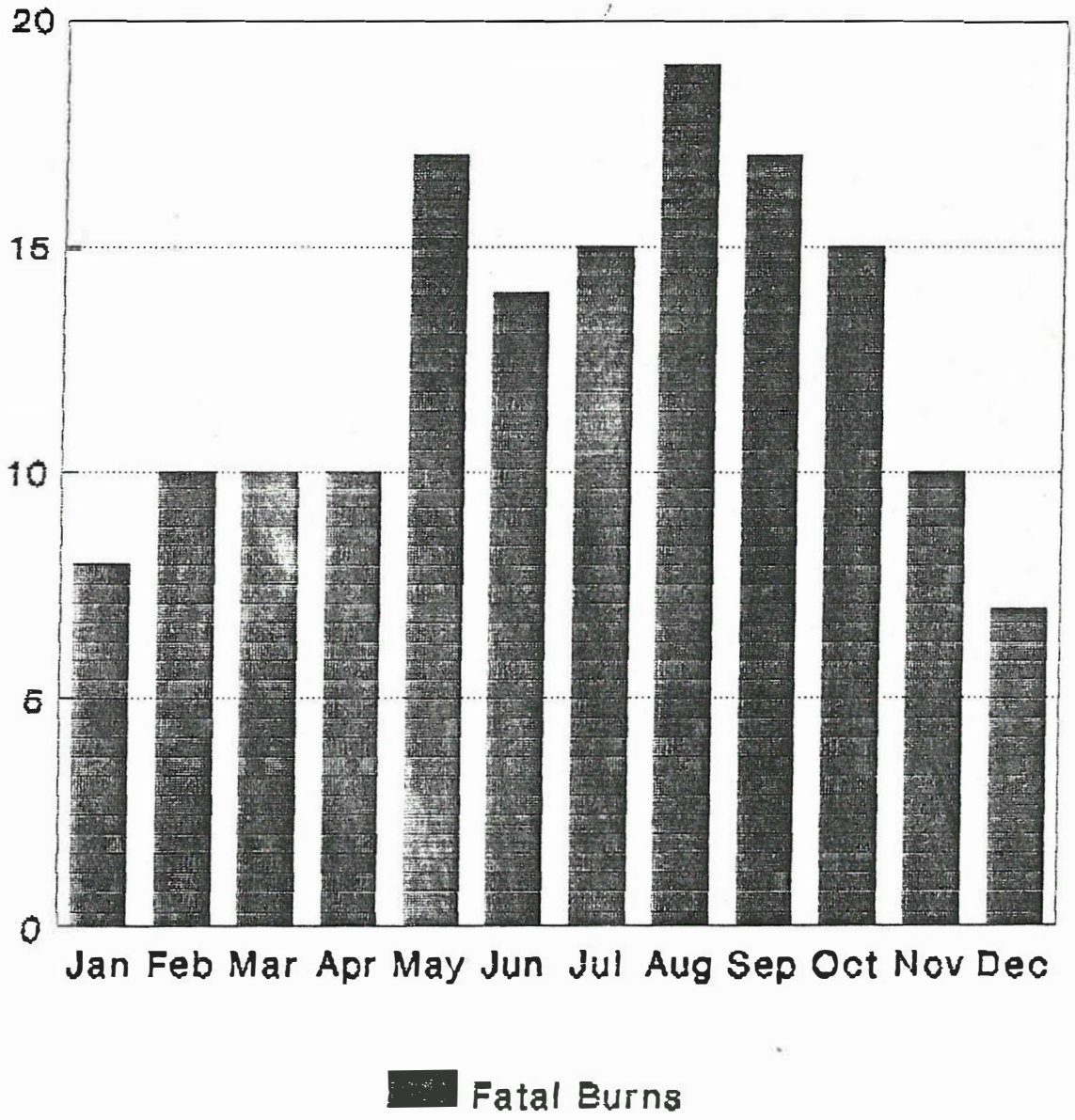


FIGURE 3.17

3.4 AETIOLOGY

Burns injury may be classified in various ways. The following classification has practical application and encompasses all the aetiological factors.

3.4.1 Classification of Burn Injury

1. Thermal burns
   a) Heat : water, fires, fireworks, flammables, e.g. paraffin, petrol, benzine, glue, etc.
   b) Cold : frost bite, N.B. Northern Natal, OFS.
2. Electrical Burns
   a) High voltage (greater than 1000 volts) : e.g. lightning, pylon voltage - direct current.
   b) Low voltage.
3. Chemical Burns
   a) Acids - Hydrochloric, Sulphuric, Hydrofluoric.
   b) Alkalis - Caustic soda.
4. Abrasive burns
   Motor vehicle collisions (motor vehicle accident), e.g. friction abrasion or burn.
5. Burns due to Explosions
   Increased incidence during times of unrest and war, e.g. petrol bombs, hand grenades, bombs, mines.
6. Burns due to Irradiation
   Microwave burns, ionising rays, e.g. Chernobyl (16).

3.4.2 Assessment of Burns

To appraise the severity of a burn injury satisfactorily with a modicum of accuracy clinically and pathologically, two principal factors are considered:

i) the percentage of total body surface burned;
ii) the depth of the burn.

Of the two, the percentage of total body surface burned is of greater significance.

Additional factors usually considered because of their influence on the prognosis are:

i) the site of the burn, e.g. facial/upper respiratory tract burns;
ii) the age of the victim;
iii) prior physical condition of the victim;
iv) concomitant injury or injuries.

3.4.3 Methods of Assessment of Burns

The assessment of the extent of burns may be categorised by one of the following means:

I Clinical
II Pathological
III Aetiological

3.4.3.1 Clinical

3.4.3.1.1 Estimation of total body surface area burned

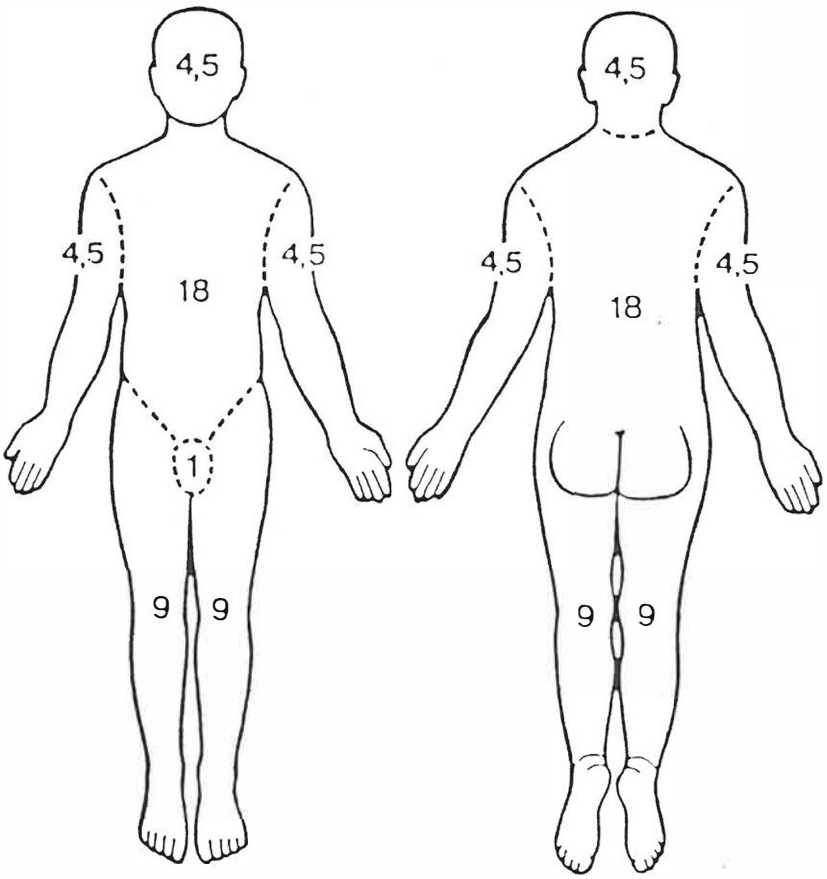
The extent of the burn is usually expressed as a percentage of the total area of the body surface that is injured.

a) Rule of Nines (Pulaski and Tennison, Wallace)

The body surface is divided into areas representing nine percent or multiples of nines (Refer to FIGURE 3.18). The Rule of Nines is reasonably accurate for clinical purposes as a rule of thumb, works well for adults but is too imprecise for children. Using this simplified rule, burns tend to be overestimated and occasionally lead to excessive fluid administration (fluid overload), which is more critical in children than in adults (14) (16).

FIGURE 3.18 BODY SURFACE AREA BURNED

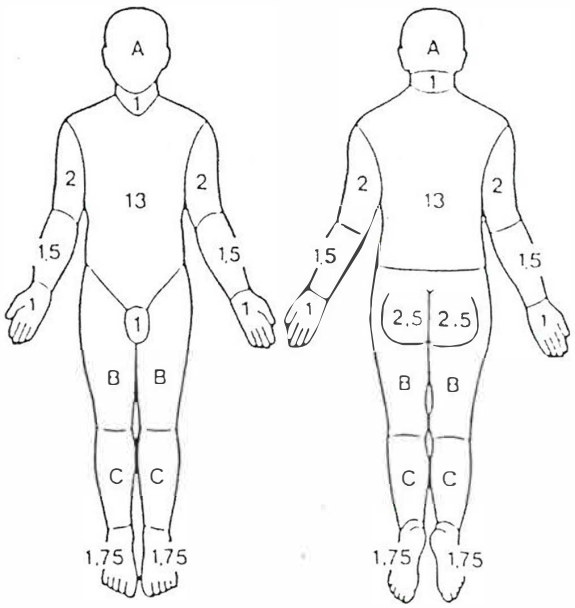
THE RULE OF NINES/ 9's



b) Lund and Browder Charts

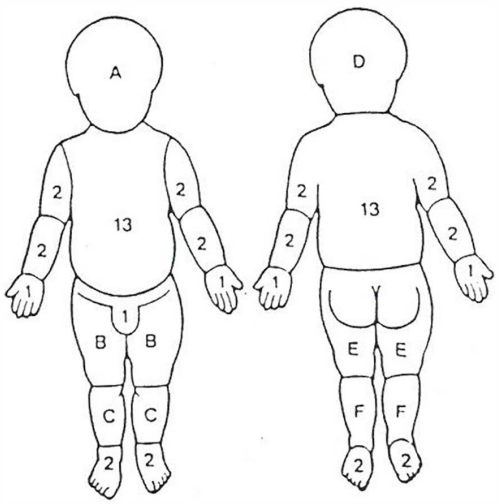
The most accurate method for determining the extent of burns is by mapping out the injury on Lund and Browder Charts (FIGURE 3.19). These are more accurate for children and adults than the Rule of Nines and are useful in both the clinical and pathological situations (14,16).

FIGURE 3.19 - LUND AND BROWDER CHARTS



AGE years	0	1	5	10	15	Adult
A = ½ of head	9.5	8.5	6.5	5.5	4.5	3.5
B = ½ of one thigh	2.75	3.25	4	4.25	4.5	4.75
C = ½ of one leg	2.5	2.5	2.75	3	3.25	3.5

Assessing the Burn
Rule of 9's too imprecise



AGE IN YEARS	0	1	5	10
Head (A/D)	10	9	7	6
Thigh (B/E)	3	3	4	5
Leg (C/F)	2	3	3	3

3.4.3.1.2 Estimation of the Depth of the Burn

Although different classifications have been used to differentiate between various depths of burns, it has been common practice to divide burns into three categories: first degree, second degree and third degree (Refer to TABLE 3.11).

a) First Degree Burn - Superficial Partial Thickness Burns e.g. scalds.

b) Second Degree Burn - Deep Partial Thickness Burns. The entire thickness of the epidermis is lost. It heals spontaneously from regeneration of the epithelium, if the wound is clean.

c) Third Degree Burn - Deep Full Thickness Burn. Both epidermis and dermis are destroyed, including dermal appendages, e.g. sweat glands and hair follicles. Skin grafting is required to ensure proper healing, failing which it will heal by contracture and scarring.

Partial thickness burns are converted to full thickness burns if allowed to dry out or become infected (14) (16).

TABLE 3.11

DIAGNOSIS OF DEPTH OF BURNS			
	FIRST DEGREE Partial Thickness (Superficial)	SECOND DEGREE Partial Thickness (Deep)	THIRD DEGREE Full Thickness
COLOUR	Red Blanches	Red-White. Does not blanch	White, Brown, Black
BLISTERS	Large	Few	None. (Dry flakes of skin)
SURFACE	Very wet	Wet	Dry
TEXTURE	Doughy	Variable	Solid
HAIRS	Attached	Variable	No hair
SENSATION TO PIN-PRICK/ LIGHT TOUCH	Present	Variable	Absent
PRESSURE AND TEMPERATURE	Present	Absent	Absent
ESCHAR AT ONE WEEK	Peeling and Healing	Level with unburned skin	Dehydrated - Sunken relation-ship to skin
HEALING	Re-epithelise in ten days	Re-epithelise in 14-35 days	Require Split-thickness skin graft

3.4.3.2 PATHOLOGICAL (Refer to FIGURE 3. 20)

3.4.3.2.1 First Degree Burns

Injury is limited to the epidermis; marked by erythema with no blister formation and is due to dilatation of arterioles and capillaries. This mild degree of burn is usually caused by sunburn, flashburn or splash burns, usually healing with no scar formation. It is equivalent to a superficial partial thickness burn in the clinical classification by depth. Pain and oedema are the chief problems. Healing readily occurs by simple desquamation. Since this is a superficial injury, the capacity of the skin to prevent infection is retained. These areas of injury are not included in the total estimation of the burnt area (14).

3.4.3.2.2. Second Degree Burns

Here there is incomplete involvement or injury to the dermal epithelial elements or epidermal destruction with necrosis, leading to separation of the dermis and the epidermis. Injury to the smaller vessels with consequent fluid loss from the intravascular compartment leads to bullae or blister formation and the development of oedema. This type of burn is usually moist and healing occurs by resurfacing from the epithelial elements which are present in the dermis. It is equivalent to a deep partial thickness burn in the clinical classification by depth (14).

3.4.3.2.3 Third Degree Burns

There is irreversible, virtually complete or near-complete destruction or necrosis of the epidermis and dermis. These burns heal by scarring and contractures, in the absence of skin grafts; such burns are prone to infection. Thrombosis occurs in the small vessels of the underlying tissue. Capillary permeability and oedema are greater in extent than in second degree burns. This is equivalent

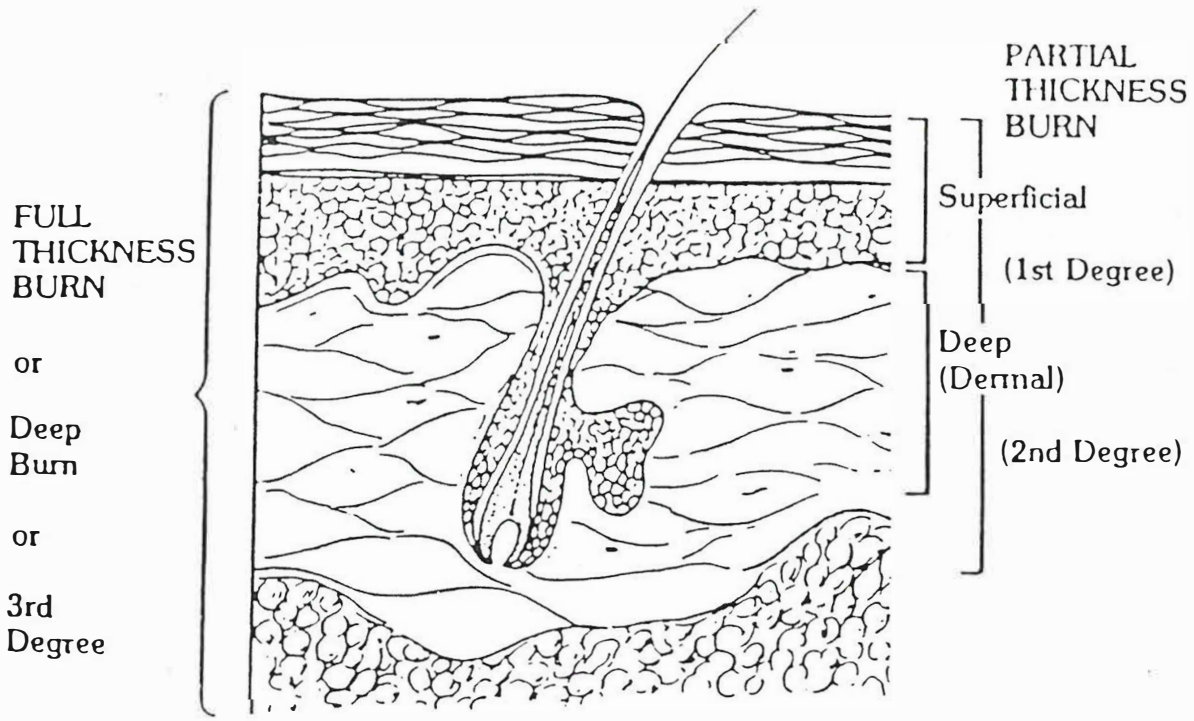
to full thickness burns in the clinical classification by depth (14).

3.4.3.2.4 Fourth Degree Burns

Charring or carbonisation of the body or relevant parts is the end-degree of burning. The skin and subcutaneous tissue are charred (i.e. black, resembling charcoal) and the muscle protein is coagulated causing muscle contraction. Charring occurs at a temperature of 300°C (15).

FIGURE 3.20 - DEPTH OF BURN

FIRST DEGREE VS THIRD DEGREE



3.4.3.3 AETIOLOGICAL

i) Accidental, e.g. children and women in the household, car catches fire in a motor vehicle collision, industrial burns, etc.

ii) Suicidal, e.g. pouring flammable liquid, usually paraffin over self and setting clothing alight, pouring boiling water over oneself.

iii) Homicidal, e.g. open flame burn, petrol bombs, victim set alight to destroy evidence-"necklace murders".

CHAPTER 3

REFERENCES

1. Haberal et al: Epidemiology of Burns in Turkey: Burns, 1987; 13 (2): 136-140.
2. Husam El Muhtaseb, Salah Qaryoute and Sameeh Abu Ragheb: Burn Injuries in Jordan Burns, 1983; 10 (2): 116-120.
3. Peter Lyngdorf: Epidemiology of Childhood Burns: Burns, 1986: 12 (4): 250-253.
4. Phillips W. et al: Epidemiology of childhood scalds in Brisbane, Burns, 1986; 12 (5): 343-350.
5. Schwär T.G, Loubser J.D, Olivier J.A. Exposure to temperature change. In: The Forensic ABC in medical practice 1st Edition Pretoria. Haum Educational Publishers 1988: 198-215.
6. R.F Brown. Injury by Burning In: J.K Mason ed. The Pathology of Violent Injury ed. London: Edward Arnold Publishers Ltd, 1978: 113-136.
7. Tedeschi C.G. Thermal Injury. In: Tedeschi C.G, Ackroyd F.W, Eckert W.G and Tedeschi L.G ed. Forensic Medcine, Volume 1, Mechanical Trauma, 1st Edition, Philadelphia: W.B Saunders 1971: 715-728.
8. Mickel R. et al: Burns Resuscitation Manual Chapter 9, N.B.T.S. Pinetown: 1-18.
9. Byron et al: Flame burn injuries. Burns, 1984; 11 (1): 1-8.
10. R.L Bang and K.M Mosbah: Epidemiology of burns in Kuwait: Burns, 1988; 14 (3): 194-200.
11. Lyngdorf et al: Burn injuries in a Scandinavian population; Burns (1986) 12 (8): 567-571.
12. Zhi-Xiang Zhu et al: Childhood burns epidemiology in China Burns, 1988; 14 (5): 394-396.
13. R.K Gupta and A.K Srivastava: Study of fatal burn cases in Kanpur, India. Forensic Science International, 1988; 37 (2): 81-89.
14. Artz C.P and Moncrief J.A (Editors). Initial Local Care. In: The Treatment of Burns. Philadelphia: W.B Saunders, 1969: 142-182.
15. Bloch, Cecil. The Burns Handbook. Pinetown; Smith and Nephew Medical Division/Lithotone 1987: 7-21.
16. Abu Ragheb Sameeh, Qaryoute Salah and Husam El-Muhtaseb. Mortality of burns in Jordan. Burns 1987; 10 (6): 439-443.

CHAPTER 4

THE PATHOPHYSIOLOGY OF BURNS

DEFINITION

A burn is a local injury caused by extremes in temperature, with an initial primary injury restricted to the surface of the body (the skin) (21).

The pathophysiology of thermal injury includes a complex set of metabolic responses that ultimately dictate the outcome of the injurious assault. The burn injury is followed by bodily adjustments both local and systemic, which influence the extent of the burn shock. Not all of these adaptions are fully understood. They include alterations in the cell membrane potentials which in turn incur physiological regulation throughout the body, including haemodynamic, acid-base, hormonal and haematological changes.

4.1 THE LOCAL EFFECTS OF BURNS

Of foremost importance is the extent and depth of the burn which are used to evaluate a burn injury. The lesion should be correctly viewed as a three-dimensional injury - this applies to all grades of burns.

A burn leads to cell injury due to tissue protein denaturation and coagulation necrosis of the underlying subcutaneous tissue. The severity of injury to the tissue is proportional to:

i) the duration of exposure to the heat source;
ii) the intensity of the temperature of the heat source;
iii) the surface area involved, and
iv) the capacity of the affected tissue to dissipate the heat (1).

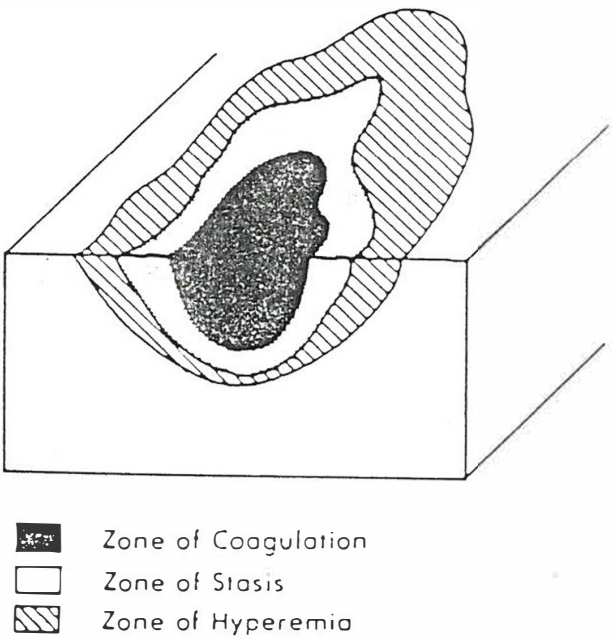
At forty to forty-five degrees centigrade, the metabolism of hyperthermal tissue is accelerated; at temperatures greater than fifty degrees centigrade, epidermal tissue may or may not develop epidermal necrosis. However, at temperatures

greater than fifty degrees centigrade, trans-epidermal necrosis occurs due to denaturation of the protein elements of the cell (1).

The burn wound consists of three zones (15) (FIGURE 4.1):

i) zone of coagulation (inner core) - necrotic, non-viable zone of tissue;
ii) zone of stasis (central) - an ischaemic zone, which may recover. However, if it becomes necrotic, sepsis oedema and shock may develop;
iii) zone of hyperaemia (outer) - inflammation tissue survives, requiring seven to ten days recovery interval.

FIGURE 4.1 - ZONES OF A BURN WOUND



The application of heat to tissue causes the release of vaso-active substances from the injured tissue and increases capillary permeability, which permits loss of fluid and protein from the intravascular into the extravascular compartment. The fluid that shifts across compartments is proportional to the extent of the burn and may appear in unburned tissue. The increased permeability also allows the egress of fluid, electrolytes, proteins, red and white cells - constituents of an exudate (2). In burns exceeding twenty-five to thirty percent of body surface area, generalised oedema or anasarca may develop caused by the increased vascular permeability. Loss of protein (globulin), leucocytes, supression of the cellular and humoral immunity render the burns victim more prone to infection at the local site and other parts of the body.

With third degree burns, the decreased rate of blood flow at the burns site is followed by stagnation, sludging and intraluminal agglutination of red cells in arterioles, venules and capillaries. Sevitt (8) showed that post-thermal induced necrosis is caused by:

a) the direct effect of the heat on the exposed tissue;
b) the concomitant stasis of blood flow causing stagnant hypoxia and hence the slough of overlying skin.

Microscopically, this is apparent as intra- and intercellular oedema of epidermal cells and the subcutaneous tissue, nuclear vacuolisation of epidermal cells, separation of epidermis from dermis and disorganisation of the dermal appendages. Further, dermal collagen fibres are separated by oedema fluid, displaying increased refractivity and loss of eosinophilia (1).

Clinically, the lesion is classically excruciatingly painful and persists for a long time, owing to the continuation of the chemical reaction, particularly in magnesium burns.

4.1.1 Bacterial Involvement of the Burn Wound

Definitions

4.1.1.1 Supra-eschar bacterial colonisation: the growth of bacterial colonies on the burn wound surface.

4.1.1.2 Intrafollicular colonisation: bacterial growth within pits of destroyed follicles which penetrate through the eschar, dermis and subcutaneous tissue.

4.1.1.3 Intra-eschar colonisation: bacterial infiltration of thermally coagulated tissue.

4.1.1.4 Burn wound sepsis: bacterial infiltration of subjacent viable tissue (Synonyms: invasion of burn wound infection) (8).

4.1.1.5 The mechanism of bacterial wound colonisation

From the first day or so post injury, the surface of the injury is generally sterile. Within forty-eight hours, bacteria colonise the wound surface, thereafter the causative agent actively invades the viable subeschar tissue with the development of bacteraemia (10). The source of the contaminant remains obscure - it may be from normal skin or colonic flora, that is, self contamination or supervening bacterial invasion. In addition, contamination from the nasal passages and hands of attendants, pose a danger. Pseudomonas aeruginosa is recognised as the most common and important coloniser, especially in burns greater than sixty percent of the body surface area (10). Pseudomonas is universally ubiquitous in distribution. Other pathogens include Proteus, Escherichia coli, Aerobacter, Staphylococci and Streptococci. Formerly Staphylococcus aureus was noted as the most common offender, followed by Gram negative organisms. More recently analyses show a rise in contamination of burn wounds by Aerobacter; Candida is frequently seen as a burn wound coloniser and systemic Candida and Mucor mycosis have been recorded in the literature, but are rare (1).

Wound sepsis can delay or prevent the formation of granulation tissue. Streptococci are known to cause grafts to slough; cellulitis and septicaemia are other complications of Streptococcal infection. Septicaemia and metastatic visceral lesions caused by Staphylococci and Gram negative organisms play a significant role in the mortality of the burns victim.

The distant septic complication due to burn wound sepsis, is primarily related to the development of bronchopneumonia, the most common complicating infection. It is postulated that bacteria are thought to become airborne or aerolise during the changing of dressings and are inhaled by the patient. Sputum cultures from patients often reveal the same causative agent as present in the septic wound. Alternatively, pneumonia is caused by bacterial invasion of the blood stream from the viable subeschar tissue. This occurs in approximately one third of burn victims (10).

Immunologically, the burns victim is compromised proportional to the extent and depth of burns - maximal in patients with burns in excess of fifty percent of total body surface area. Compliment abnormalities, hypogammaglobulinaemia, depressed humoral and cell mediated immunity, decreased neutrophil intracellular bacterial killing and abnormal inflammatory response within the burn wound, have all been described in the literature. Neutrophil and monocytic chemotactic responses are also markedly decreased (2). The post-injury immunosuppression predisposes the burn victim to a higher than normal incidence of invasion by opportunistic bacterial, fungal or viral organisms (2).

4.2 THE SYSTEMIC EFFECTS OF BURNS

The deleterious effects of burns on the various systems of the body and the extent and duration of organ dysfunction are proportionally related to the extent and depth of the burn. The systems predominantly affected following thermal injury include the cardiovascular, pulmonary and urogenital systems.

The major cause of "burn shock" is the increase in the capillary permeability which occurs during the first twelve to thirty six hours. Other factors contributing to the development of the hypovolaemic shock are the increased evaporative water loss, increased fluid sequestration in muscle cells, and less significanty, red cell destruction (15).

In burns involving less than thirty percent of total body surface area, fluid extravasates into the tissues, causing local oedema. With larger burns, involving more than thirty percent of total body surface area, the loss of fluid into the extravascular compartment is generalised (generalised oedema). The latter causes severe reduction in circulating fluids and consequent hypovolaemic shock. Hypotension mediated in all probability by the release of metabolites from the arachadonic acid cascade, consisting of Prostaglandins E_4 and F_4, Prostacyclin and leukotrienes C_4, D_4 and E_4. (15).

Another significant factor in the development of burn shock is the loss of water from the burn wound. Evaporative water loss can reach peak levels of up to 350mℓs per hour or 0,35mℓs per square centimetre of burn area per day (15,16).

The increased permeability of capillaries is not limited to fluid only, but also to protein molecules, into the interstitial space. This further accentuates both the tissue oedema and the vascular hypoproteinaemia. During the first ninety-six hours post burn, albumin lost from the circulation is equal to twice the total normal plasma pool in the body. (15)

At the cellular level, the membrane potential of the cell is altered, the malfunctioning sodium-potassium pump leading to increased intracellular sodium and water, while the potassium level in the extracellular compartment is elevated (15).

All the preceding events lead to a reduced circulating fluid volume which induces aldosterone release, that increases urinary potassium excretion; sodium is conserved by the presence of circulating anti-diuretic hormone. Shock is further aggravated by the "myocardial depressant factor", which decreases cardiac

output in large burns (greater than sixty percent of total body surface area), and the release of catecholamines, which increase the peripheral vascular resistance (4).

Initially, haemoconcentration occurs with an elevated haemoglobin and haematocrit. An increased viscosity and enhanced platelet stickiness occur, causing sluggish blood flow and aggregation of platelets. A ten to fifteen percent destruction of red blood cells is directly caused by the thermal injury, while the life of the remaining blood cells is decreased by ten to twenty-five percent, leading to anaemia (15). The free plasma haemoglobin released is rapidly excreted by the kidneys where precipitation of the haemoglobin leads to the formation of obstructive casts. Tubular damage caused by prolonged shock with peripheral collapse and obstructive casts, lead to acute renal failure or acute tubular necrosis (16).

A metabolic acidosis often occurs while decreased gastric motility, peripheral and splanchnic vasoconstriction lead to an ileus and gastric ulceration. (15)

In summary, the burns victim undergoes burn shock due to hypovolaemia which manifests in virtually every system in the body. There is a reduced urine output and initial rise in haematocrit and haemoglobin. Untreated, the patient will develop acute renal failure or acute tubular necrosis. The hypovolaemia and decreased blood flow lead to ischemia and hypoxia to the brain, clinically manifested by restlessness (15).

CHAPTER 4

REFERENCES

1. Tedeschi C.G, Ackroyd F.W, Eckert W.G. and Tedeschi L.G. ed. Forensic Medicine, Volume 1, Mechanical Trauma, 1st edition. Philadelphia: W.B. Saunders, 1977: 715-728.
2. Pruitt B.A. and Goodwin C.W. Burns. In: David C. Sabiston, ed. Textbook of Surgery 12th edition Philadelphia: W.B. Saunders, 1981: 287-316.
3. Aikwana N. et al. Multiple organ failure in burned patients. Burns, 1987; 13 (2): 103-109.
4. Robbins S.L, Cotran R, Kumar V. Pathological Basis of Disease. 3rd edition. Philadelphia: W.B. Saunders, 1984: 114, 1026-1035.
5. Herndon D. et al. Respiratory injury, Part 1. Burn Care and Rehabilitation, 1986; 7 (2): 184-195.
6. Schiavon M. et al. Renal damage in severely burned patients. Burns, 1988; 14 (2): 107-114.
7. Anderson J.R. The Urinary System. In: J.R. Anderson, ed. Muir's Textbook of Pathology. 11th edition. London: Edward Arnold, 1985: 138-145.
8. Teplitz C. The Pathology of Burns. Artz C.P. and Moncrief J.A, ed. The Treatment of Burns. 2nd edition. Philadelphia: W.B. Saunders, 1969: 22-28.
9. Chen Yi-Sheng, Davies J.W.L. et al. Histopathological and ultra-structural changes in liver tissue from burned patients. Burns 1985; 11 (6): 408-418.
10. Curreri P.W. Burns. In: Schwartz S.I. Principles of Surgery: 3rd edition. New York Mc Graw Hill: 286-301.
11. Wartman W.B. Mechanisms of Death in Severe Burn Injury. In: C.P. Artz, eg. Research in Burns. 9th edition. Washington: American Institute of Biological Sciences, 1962: 6-14.
12. Wallner S.F. and Warren G.H. Haematopoietic response to burning. Burns, 1985; 12 (1): 22-27.
13. Pruitt B.A. and Goodwin C.W. Burns. In: David C. Sabiston, ed. Textbook of Surgery 12th edition. Philadelphia: W.B. Saunders, 1981: 287-316.
14. Settle J.A.D. Burns, the first forty-eight hours. Hertfordshire: Smith and Nephew Pharmaceuticals, 1974: 23-24.

15. Bloch Cecil. Burns Handbook. Pinetown: Smith and Nephew Medical Division, 1987: 7-33.

16. Hummel R.P. Clinical Burn Therapy. 1st edition. Massachusetts: John Wright, 1982: 411-428.

17. Scotti T.M. and Hackel D.B. Heart. In: Kissane J.M. ed. Anderson's Pathology. 8th edition. S.E. Louis; C.V. Mosby Co. 1985: 630-631.

18. Chen Yi-Sheng, Davies J.W.L. et al. Histopathological and ultra-structural changes in liver tissue from burned patients. Burns, 1985, 11(6): 408-418.

19. Prasad J.K., Thomson P.D. and Feller I. Gastrointestinal haemorrhage in burn patients. Burns, 1987; 13(3): 194-197.

20. Mickel R., Lloyd D, et al. Burns Resuscitation Manual Chapter None. Pinetown: Natal Blood Transfusion Service: 1-18.

21. Schwär T.G., Loubser J.D., Olivier J.A. Exposure to Temperature Change. In: The Forensic ABC in Medical Practice. 1st edition. Pretoria: Haum Educational Publishers, 1988: 198-215.

22. Kinsella J. Smoke Inhalations. Burns, 1988; 14(4): 269-279.

23. Prien T. and Traber D.L. Toxic Compounds and inhalational injury - a review. Burns, 1988; 14(6): 451-460.

24. Miyoshi K., Tsukada S., Yasuda Y. et al. Hepatic disorder in burn patients. Burn, 1985; 12(1): 49-53.

25. Davies M.R.Q., Cywes S., Davies D. A Review of Deaths in a Paediatric Burns Unit. South African Medical Journal, 1976; 50: 1479-1483.

26. Haberal M., Oner Z., Bayraktar U., Bilgin N. Epidemiology of Burns in Turkey. Burns, 1987; 13(2): 136-140.

27. Abu Ragheb Sameeh, Qaryoute Salah and Husam El-Muhtaseb. Mortality of Burns in Jordan. Burns, 1987; 10(6): 439-443.

28. Bang R.L. and K.M. Mosbah. Epidemiology of Burns in Kuwait. Burns, 1988; 14(3): 188-193.

CHAPTER 5

THE PATHOLOGY OF BURNS

In fatal burns, there are usually multiple factors contributing to death. Hence "the pathologist cannot always state conclusively what a patient dies from but can only indicate what he dies with" (Teplitz) (8). With multiple mechanisms operative, which leave few, if any, anatomical traces, the cause of death is not always obvious at post mortem. During the first week after sustaining burns, most patients die of inhalational injury or shock. During week two, shock and sepsis claim most victims' lives, while during the third to fourth weeks, sepsis is the most common cause of death. In the tenth to twelfth week, serum hepatitis (Hepatitis B) accounted for most deaths. The pattern is very similar in most studies (11). The incidence of iatrogenic hepatitis has been reduced with testing of all donated blood for the hepatitis virus and also greater precaution being undertaken by both medical and paramedical staff. The minimisation of the use of fresh frozen plasma in the treatment of burns is recommended to prevent the transmission of transfusion hepatitis, especially non A, non B hepatitis (24).

The understanding of the pathophysiology of burns shock and fluid replacement therapy have contributed to the decrease in the incidence of death in the early phase of severe burns. Unfortunately, however, the patient with extensive burns survives the initial shock period, only to develop infection or organ(s) failure later in the course of their treatment. When several organs are simultaneously involved, the mortality becomes very high; the most frequently affected organ is the lung followed by the heart, kidney, liver and the blood clotting system (3).

5.1 THE RESPIRATORY SYSTEM

5.1.1 Inhalational Injury

Inhalational injury refers to the pathophysiological events involving the respiratory and circulatory systems, that may occur on exposure to fire and its products. The exact and full extent of the injury remains incompletely understood and is

a phenomenon of considerable clinical and pathological significance.

5.1.1.1 The Diagnostic Criteria for Inhalational Injury are:

i) facial and peri-oral burns with singed moustache, nasal hair, eyebrows, eyelashes and blistered lips and face;
ii) history of burn in a closed environment;
iii) respiratory signs and symptoms (wheeze, crepitations, cough, hoarseness and tachypnoea);
iv) elevated carbon monoxide levels;
v) decreased arterial oxygen levels;
vi) history of (associated with) consumption of alcohol;
vii) combustion of modern household furnishings - the type of material being burnt is relevant to the amount of damage caused;
viii) altered level of consciousness;
ix) carbonaceous sputum.

5.1.1.2 Incidence

Inhalational injuries manifest in eleven to thirty-three percent and up to fifty percent of burn victims (5)(29).

The frequency of pulmonary complications as a cause of death in patients in thermal injury has been reported as varying between 18 and 84% (5)(30)(31). In the present series this frequency was noted at 43%.

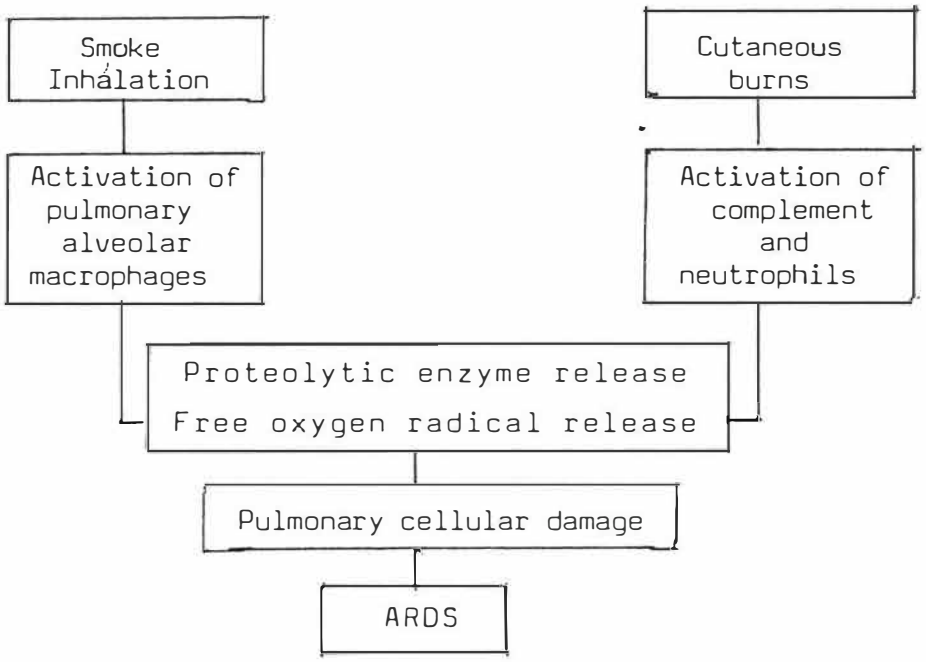
5.1.1.3 Mortality

The mortality in patients who sustained inhalational injury is uniformly reported between forty-seven and eighty-four percent (5). When comparing the incidence of mortality related to age, the proportion of patients with inhalational injury increases with increasing age; in patients older than sixty years, the mortality increases to a hundred percent (5). The elderly sustain inhalational injury more frequently than the young, probably due to their sedentary life-style. The mortality in inhalational injury with small areas of thermal injury is rather high, at thirty-six percent (5).

TABLE 5.1 - TOXIC GASES AND VAPOURS PRODUCED BY BURNING MATERIALS

TOXIC GAS OR VAPOUR	MATERIALS
CO, CO_2	All combustibles containing carbon
NO_x	Cellulose, polyurethanes, acrylonitrile, high temperature combustion of all materials
HCN	Wool, silk, N-containing plastics
H COOH, CH_3, COOH	Cellulosic materials, rayon
Acrolein	Wood, paper, upholstery, carpeting
SO_2	Rubber, thiokols, acrylics, aircraft windows
Halogen acids	PVC, many fire-retarded plastics
NH_3	Melamine, nylon, urea-formaldehyde
Aldehydes	Phenol-formaldehyde, wood, nylon, polyester resin
Benzene	Polystyrene
Phenol	Phenol-formaldehyde
P compounds	Some fire-retarded plastics containing P compounds

FIGURE 5.1 - Proposed mechanisms for the development of the Adult Respiratory Distress Syndrome in victims of smoke inhalation and cutaneous burns



In summary, the pathophysiology of the inhalation syndrome is related to a combination of heat, smoke and particulate inhalation. The presence of a surface body burn significantly aggravates the condition, the clinical course and prognosis. The clinical course is related to the severity of the injury, the response of the patient to that injury and is influenced by pre-existing cardiopulmonary disease, when present. The upper airway is injured by heat with the development of supra-glottic oedema and airway obstruction, which usually occurs within twenty-four hours. The lower airway is injured by heat, smoke and particulate matter with subsequent small airway obstruction and alveolar damage and resultant pulmonary collapse, oedema and bronchopneumonia (16).

Refer FIGURE 5.2

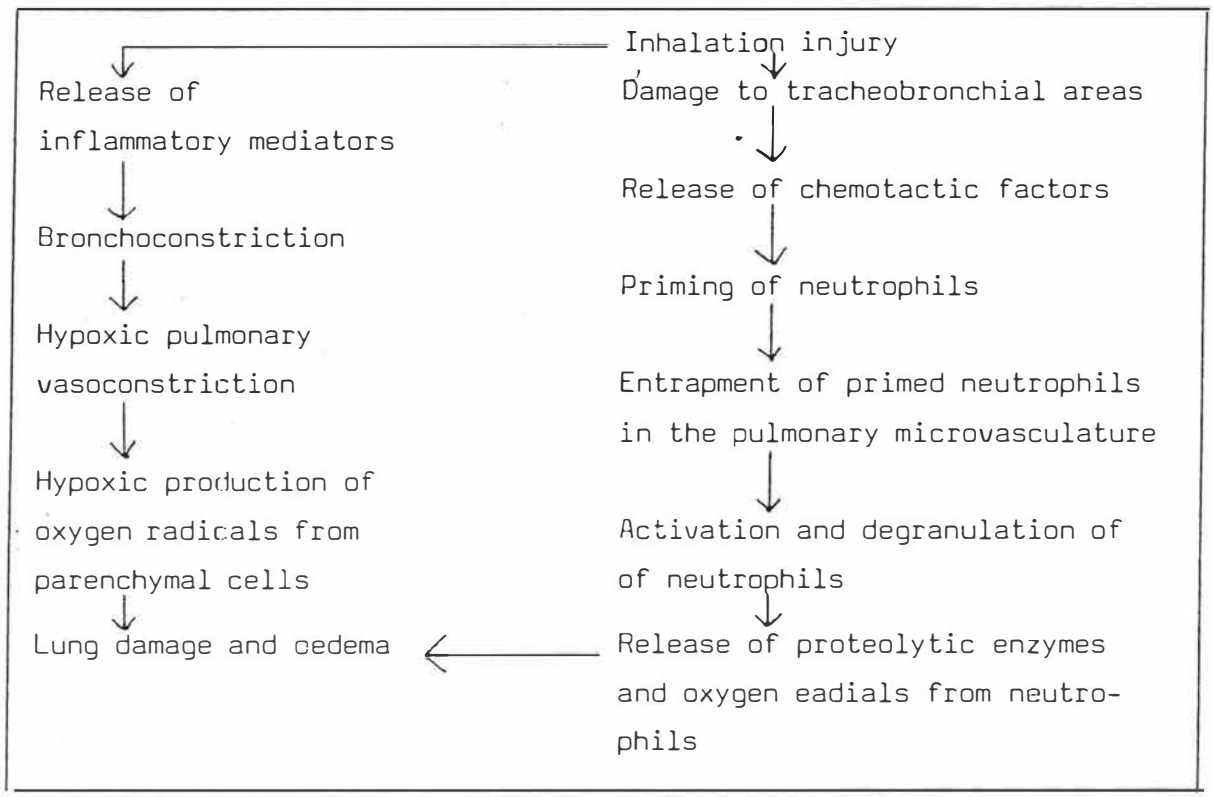
As combustion progresses, oxygen levels decrease and hypoxia is the mechanism of death at the place of fire. The increased CO_2 stimulates respiration which leads to further inhalation of smoke. With prolonged exposure, loss of consciousness and carbon dioxide retention occur (22).

Carbon monoxide is formed due to incomplete combustion of carbon-containing matter. Carbon monoxide has a high affinity for haemoglobin and readily crosses the alveolar and endothelial membranes; in addition carbon monoxide inhibits carbonic anhydrase leading to cytotoxic/histiotoxic hypoxia. Cyanide affects cellular metabolism and hence will aggravate the metabolic acidosis induced by hypoxia. Cyanide poisoning does not occur in house fires in the absence of carbon monoxide poisoning, due to variable substances burned in each fire and the elimination patterns of these substances (22). Particulate matter in smoke has a triple effect:

i) it acts as an airway irritant;
ii) it induces thermal damage when hot, especially in the lower airways;
iii) carbon particles become coated with gases containing irritant aldehydes and organic acids which are injurious to the upper and lower respiratory tract (23).

Inhalational injury causes diffuse tracheobronchitis, with necrosis and shedding of the tracheal and bronchial epithelia, occurring twelve to seventy-two hours after injury. Pulmonary oedema, a hallmark of inhalational injury, is probably mediated by the products of activated neutrophils which give rise to the "shock lung" (ARDS, burn lung) syndrome which is schematically represented in FIGURE 5.1

FIGURE 5.2 - Schema for the hypothetical sequence of events leading to parenchymal damage occurring following inhalation injury



5.1.1.5 Findings: Inhalational Injury

The diagnostic criteria were divided into clinical and post-mortem findings, in all hospitalised patients. A diagnosis of inhalational injury was entertained if two or more criteria were noted to be positive, in each group of criteria. This group consisted of a total of one hundred and thirty three patients or 88% of all fatalities.

Nineteen patients or 14,3% of all hospitalised patients had evidence of inhalational injury; of the remaining eighteen patients who had died prior to reaching hospital, five showed macroscopic evidence of inhalational injury, but could not be included as the clinical criteria were not fulfilled.

All cases that were classified as positive for inhalational injury, were checked against both clinical and macroscopic findings and when available confirmed histologically. Five victims with inhalational injury had clinical and macroscopic evidence of adult respiratory

distress syndrome. Histology was available on only two of the five and was negative in both cases.

Clinically and macroscopically nine patients were suspected of having developed adult respiratory distress syndrome. Histologically, three of these patients did not show features of adult respiratory distress syndrome, while the histological tissue was not available for examination on the other six cases. In addition, thirty five cases, representing a cross section of the total did not show any features of adult respiratory distress syndrome. However other respiratory complications were present.

5.1.2 Pneumonia in Burns

The post-resuscitation period is characterised by pulmonary dysfunction, inflammation and sepsis, with the onset of bronchopneumonia, usually forty-eight hours after injury. Impaired lung immunity and the burn-induced immunosuppression, renders the patient susceptible to circulating endotoxin, vaso-active mediators and adult respiratory distress syndrome (burn lung) and bronchopneumonia.

The pneumonia-afflicted burns victim may suffer from the following:-

5.1.2.1 Bronchopneumonia

This condition results from haematogenous bacterial spread from infected burn wounds or the tracheostomy site; with tracheostomoties the infection originates in the peribronchial tree with a rapid tendency towards confluence, in the absence of treatment, obliterating functional pulmonary parenchyma.

5.1.2.2 Hypostatic pneumonia

This is a fatal process comprising of septic, subpleural, peripheral, metastatic abscesses, secondary to bacterial spread from infected wounds. Microscopically, the capillaries are engorged with organisms and mortality is usually one hundred percent (8). In this series, thirty-nine percent of victims developed bronchopneumonia, confirmed histologically and lead to the demise of the patient.

5.1.2.3 Findings: Pneumonia in Fatal Burns

Bronchopneumonia remains the most frequent complication in hospitalised burn patients (26) (25). The results in this series confirm this well documented complication. Macroscopically, at post-mortem, thirty-nine percent or 52 hospitalised patients were diagnosed as having bronchopneumonic changes. The lungs were noted to be congested, oedematous, with or without fibrinous pleural exudate. On palpation shotty foci around bronchioles were noted, purulent exudate present in the bronchi with increased friability of the lung parenchyma. The patches of infective changes were bilateral, initially basal and were seen to coalesce into large foci (confluent bronchopneumonia).

When detailed further 39% or fifty-two hospitalised patients had macroscopic bilateral pulmonary infective changes. Confluent bronchopneumonia was noted in 25,6% or thirty-four patients, whilst in 13% (18 cases) the infection was recorded as being basal in distribution. Later as the infection coalesces, it becomes impossible to differentiate whether the initial site was basal, from a tracheostomy or haematogenous.

Diligent scrutiny of 86 patients admitted to King Edward VIII Hospital, demonstrated only three documented positive blood cultures in the group that developed bronchopneumonia. In one Pseudomonas, staphylococcus in the second and in the third isolate, Pseudomonas and Klebsiella pneumoniae were cultered from blood. Precise record of when respiratory infection had set it in was not documented and hence how long after the burns were sustained, infection had occured, could not be ascertained.

Abscess formation in bronchopneumonia is commonly associated with staphylococci and Pseudomonas. In this series seven (5,3%) hospitalised patients had naked eye evidence of pulmonary abscess development. Unfortunately pus swabs were not taken and hence the causative organism not identified.

Sepsis as reported by authors elsewhere account for more than fifty percent of deaths, associated with burns (28) (27) (26) (25) (1).

5.1.2.4 Aspiration pneumonia

Aspiration of gastric contents (usually after a meal), or infected material leads to haemorrhagic oedema and adult respiratory distress syndrome. Death can be immediate, due to obstruction, or delayed, following development of adult respiratory distress syndrome and respiratory failure (16).

5.1.2.4.1 Findings: Aspiration pneumonia

In the present local series no cases of aspiration pneumonia were identified. The criteria for diagnosing the condition were as follows:-

(i) clinical history of aspiration (if available)

(ii) macroscopic evidence of aspiration

- (a) tracheobronchitis with pink frothy luminal fluid
- (b) gastric contents in trachea or bronchi and bronchioles
- (c) microscopic evidence of aspiration
  - (a) neutrophilic reaction
  - (b) focal bronchial mucosa necrosis
  - (c) foreign body with surrounding vital reaction
  - (d) foreign body giant cell reaction
  - (e) vegetable matter with vital reaction

No history of aspiration had been forthcoming in any of the cases; although, cases of erosion and tracheitis were seen, no microscopic evidence of aspiration was noted in thirty five of the cases examined, histologically. The final arbitrator in positive macroscopic cases, was the absence of microscopic criteria.

5.1.3 Pulmonary collapse

The incidence of pulmonary collapse in one series by Teplitz (1962) (8) was thirty percent, and, of these, forty-three percent were noted to have a basal distribution. Peculiarly, bronchial or bronchiolar obstructive mucous plugs were conspicious by their absence, macroscopically and microscopically, in the same series (8). Associated congestion leads to the term "congestive atelectasis", where the collapsed areas appear dark red to violet in colour, and are filled with blood; the pleural surfaces have no depressed areas and hence resemble foci

of consolidation. This condition is more common in children, and a restricted mechanical expansion of the thoracic cage has been postulated as a cause.

5.1.3.1 Findings: Pulmonary collapse

Pulmonary collapse was noted macroscopically in three cases but could not be confirmed microscopically nor did it feature in the thirty-five cases examined histologically.

Intrapulmonary haemorrhage was reported macroscopically in eight hospitalised cases; in seven the haemorrhage was distributed focally and only in one case was it bilateral and diffuse.

An additional three cases with intrapulmonary haemorrhage were present in the group that died prior to reaching hospital.

5.1.4 The Larynx and Trachea

Focal erosions of the laryngeal and tracheal mucosa were common in a series by Teplitz (1962), especially in those patients who survived for at least three weeks. Seventy five of these cases showed laryngeal erosions, while patients with tracheostomies commonly developed pressure erosion on the anterior wall of the trachea, at the abutment site of the tracheostomy cannula tip The erosions occurred in the absence of inhalational injury. These lesion are often colonised by bacteria indigenous to the burn wound. Haemorrhage with consequent aspiration is a possible fatal complication; it however does not appear to be a common complication (18) (16).

5.1.4.1 Complications of Tracheostomies

In burns patients, where relatively prolonged mechanical ventilatory support is required, elective tracheostomy is preferred to endotracheal intubation. However, tracheostomies are not free of complications even with careful attention, for example:-

i) anterior mucosal ulceration of lower trachea in contact with the tip of the catheter;
ii) predisposition to invasive respiratory infection;

iii) tracheal perforation;
iv) bronchospastic respiratory obstruction, especially in children;
v) pulmonary collapse, particularly with low tracheostomies (8).

5.1.4.2 Findings: Larynx and Trachea

The mortality due to pulmonary complication in this study was in keeping with the trend described by authors in the earlier review.

Pulmonary complications were responsible for forty-three % of deaths in this study as corroborated by other series (28), (27), (26), (25), (1), where pulmonary complications have replaced shock as a major cause of mortality in burned patients.

5.1.4.3 Findings: Tracheal Ulceration, Tracheitis

Tracheal and laryngeal ulceration was present in 7,5% (10 patients) of the hospitalised cases. The erosions presented in the absence of inhalational injury. Erosions usually involved the laryngeal mucosa, especially in intubated patients. The tracheal ulcers were located on the anterior wall in both intubated and non-intubated patients.

Tracheitis was present in significantly more victims 35 patients or 26% of the hospitalised victims.

In two patients, (1,5%) haemorrhage was present but in neither case was it life threatening or fatal, in consequence.

5.2 THE CARDIOVASCULAR SYSTEM

Early in the post-burn period, the cardiac output decreases due to an increased peripheral resistance, decreased blood volume and increased viscosity; the latter is caused by an increased hematocrit and aggregation of red cells, platelets and white cells (2).

5.2.1 Myocardial Depressant Factor

A circulatory cardiotoxic factor, named myocardial depressant factor (MDF), which is a pancreatic polypeptide, has been identified in burns

involving greater than sixty percent of the body surface. It is released from the ischaemic pancreas and has a direct depressive effect on myocardial function, causing acute myocardial insufficiency, and occasionally, cardiac arrest in the burns patient (4). It is hypothesized that the cardiac effects of MDF may be related to the hyperkalemia, that originates from the release of intracellular potassium from the damaged and haemolysed erythrocytes, and also excessive catecholamines (17).

5.2.2 Catecholamines

The hypermetabolic response in burns appears to be a catecholamine cascade, due to "resetting" of the central hypothalamic - adrenal axis. This central stimulus remains unknown. Catecholamines enhance myocardial oxygen consumption and are though by many authors to induce hypoxic injury, when in excess, causing exhaustion of available oxygen to the heart via the coronary arteries, and manifesting as subendocardial haemorrhage (17).

Myocardial infarction, a rare complication of burn shock and injury, is seen more commonly in the elderly.

5.2.3 Thrombo-embolism

The burn patient is the ideal candidate for thrombotic complications, but the autopsy findings of published reports show this to be rare, except perhaps in the elderly. More recent impressions by some authors is that the incidence seems to be increasing, although the exact incidence is not known (16).

5.2.4 Findings: The Cardiovascular System

Myocardial infarction did not feature in any of the victims of burns, who were admitted. Clinically no patient in the series was diagnosed as having had a myocardial infarction. Macroscopically no evidence was obvious at necropsy and no microscopic evidence ofinfarction recent or healing,was seen on the group of cases examined histologically.

Catecholamine cardiotoxicity was examined for by examining for subendo-

cardial haemorrhage (SEH) of cases. SEH was present in 13,5% or 18 victims. SEH remains a good indicator of hypoxic myocardial injury as in shock.

The criteria for the diagnosis of shock in this series were as listed:-

(i) prolonged decrease in blood pressure or central venous pressure with consequent hypoxia
(ii) Lungs: Adult Respiratory Distress Syndrome
(iii) Heart: Subendocardial Haemorrhage
(iv) Gastrointestinal Tract (GIT)
  (a) GIT ulceration
  (b) Focal hepatic hypoxia/necrosis
  (c) Focal Pancreatic Necrosis
(v) Brain: "Watershed" infarcts
(vi) Adrenal Haemorrhage
(vii) Disseminated Intravascular Coagulation
(viii) Acute Tubular Necrosis (see below)

The post-mortem changes in shock are not striking and organ damage described above may be present in varying degree and combination.
A diagnosis of shock was considered when at least four criteria were satisfied.
In this series, eight victims out of a total of one hundred and fifty one (5,3%) victims were diagnosed as having shock.
Alternatively, 6% of hospitalised patients developed shock, using the above criteria.

The criteria used for the diagnosis of pulmonary embolism, in this series, was the macroscopic presence of pulmonary emboli. Five patients or 3,8% of hospitalised victims succumbed to this fatal complication.

5.3 THE KIDNEYS

After the lung and heart, the kidney is the next most frequently affected organ in burn victims (3). Advances in the understanding of the pathophysiology of burn shock and the treatment thereof have led to a decrease in mortality due to renal failure.

5.3.1 Acute Renal Failure (Acute Tubular Necrosis - ATN)

5.3.1.1 Definition

All forms of renal failure associated with the destruction of tubular epithelial cells (4).

In burns, the ischaemic type of acute tubular necrosis occurs after an episode of shock complicated by peripheral circulatory collapse (4).

ATN is a reversible disorder compatible with full recovery of renal lesions and of the patient if treated timeously and adequately.

5.3.1.2 Incidence

In the severely burned patient, 1,3 to 38% of victims develop ATN (6). In this series, seventeen percent of victims had evidence of acute tubular necrosis.

5.3.1.3 Mortality

The reported mortality of burns patients who develop ATN is high a 73 to 100% (6). (With the onset of renal failure in burns, mortality usually tends to be very high).

5.3.1.4 Aetiology

The aetiology of ATN is multi-factorial and the precise mechanisms are not fully understood, but the following factors do play a role:-

(i) shock with impaired renal blood flow;
(ii) peripheral circulatory collapse (4);
(iii) a distal tubular lesion thought to render it refractory to aldosterone (6);
(iv) a proximal tubular lesion with a large load of non-resorbable solutes causing an asmotic diuresis (6);
(v) free haemoglobin derived from haemolysed erythrocytes and muscle injury accentuate cast development.

5.3.1.5 Occurrence

The condition is usually seen 2 to 3 weeks after burn injury manifesting clinically as oliguria or anuria, with increasing blood urea and creatinine levels.

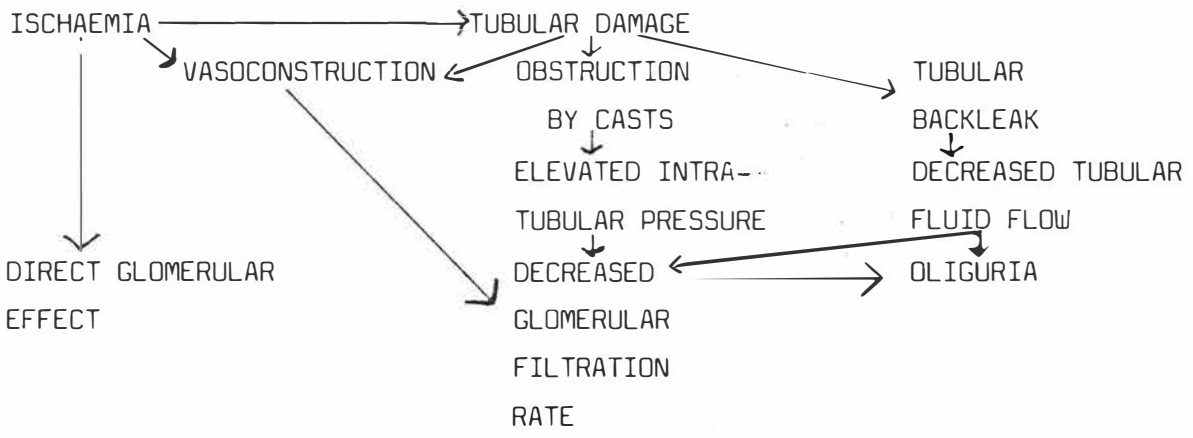
5.3.1.6 Pathophysiology of ATN

Thermal injury leads to a prolonged shock with hypovolaemia, hypotension, decreased cardiac output, increased peripheral resistance, hypoproteinaemia and with the addition of circulatory collapse sets the stage for ATN. Deposition of pigment casts in the medullary tubules is considered by some authorities as the most pathogenetic factor causing ATN. The casts cause intrarenal obstruction which leads to microcirculatory insufficiency and terminally to renal ischaemia and acute renal failure. In addition, urinary granular fragments, epithelial cells and red blood cells from casts (4).

5.3.1.7 Pathogenesis of ATN

FIGURE 5.2 represents a schematic diagram of the pathogenesis of ATN

FIGURE 5.3 - POSSIBLE PATHOGENETIC MECHANISM IN ACUTE TUBULAR NECROSIS



5.3.1.8 Pathology of ATN

Macroscopically, the kidneys are enlarged (nephrogemaly), the cortex appears swollen and pale or streaked because it contains little blood and occasionally may show petechial haemorrhage. The medulla appears congested and dark (7). The dilated tubules are lined by flattened epithelium, when not necrosed. The type of cast may be granular, protein, hyaline and epithelial in type located in the distal tubules and collecting ducts. All casts comprise of Tamm-Horsfall protein.
Tubular regeneration and extramedullary haemopoesis are occasionally seen.

In the histological analysis of ATN, extra caution should be exercised as the condition closely mimics post-mortem autolysis.

TABLE 5.2 - HISTOLOGICAL APPEARANCES ON LIGHT MICROSCOPY IN ACUTE TUBULAR NECROSIS

Bowman's capsule	Normal
Glomeruli	Normal
Proximal tubules	Often normal, but epithelial change due to mannitol may be present
Distal tubules	Focally dilated and lined with flattened epithelium. Pigmented casts are prominent and tubular necrosis, if present, is usually where a cast is located
Vessels	Normal
Interstitium	Slight focal oedema and focal infiltration of lymphocytes, plasma cells and granulocytes

5.3.1.9 Findings: Acute Tubular Necrosis

The diagnosis of ATN in this series, as elsewhere, was based on six macroscopic and four microscopic criteria, outlined earlier (4). In view of the prolonged post-mortem interval autolysis was a recurrent problem and therefore on no occasion were all the criteria met. Microscopically the presence of intraluminal tubular casts was the most significant criterion microscopically and in addition three macroscopic criteria were essential for positive assessment : nephromegaly, pale swollen cortices and congested medullae. Macroscopically 65 victims

(49%) had demonstrated features suggestive of ATN. Histological confirmation on a representative sample showed that 17% of burn victim had features of ATN. The inference can be made that the macroscopic appearance (49%) and microscopic appearance do not demonstrate a good correlation.

5.3.2 Cystitis

The occurrence of cystitis is predisposed by the presence of indwelling catheters and bacteriuria. The bacterial flora in the urine is identical to that colonising the burn wound. The problem can be minimised by frequent irrigation and renewal of indwelling catheters every five days (8).

5.3.2.1 Findings: Cystitis

The type of cystitis usually encountered in the burn victim is the haemorrhagic type. In this series 30% or forty hospitalised patients showed macroscopic evidence of haemorrhagic cystitis. Histology for confirmation was not available.

5.4 THE LIVER

The liver changes in burn victims are non-specific responses to injury. The impairment of liver function in burns is probably due to hypovolaemia with inspissation of bile secreted during the first few days in the post burn period (9).

Macroscopically, hepatomegaly, congestion and a fatty liver may be apparent. Histologically, the following were noted in a recent review (Davies et al) (18).

a) centrilobular fatty change was the most common finding;
b) degeneration and necrosis of hepatic cells;
c) proliferation of Kupffer cells showing active phagocytosis. It is probably a compensatory reaction to the severe splenic injury;
d) necrotic cells in hepatic sinusoids;
e) hepatic cell regeneration.

Most histological sections showed gross abnormalities, in this series, with normal architecture a rarity in severely burned patients. Hepatic necrosis is the end result of prolonged hypoxia due to circulatory insufficiency of greater than twenty-four hour duration (16). Hepatic regeneration begins after one week, if the patient survives. In contrast, the findings by Teplitz (1979) and Sevitt (1957) are only slightly different and the divergent nature of these results may be partially explained by the period between the time of burn and death which was not reported.

5.4.1 Findings: The Liver

In this series, macroscopically and microscopically, the following were examined:-

(a) Fatty change:-
  (i) extent (mild to moderate to severe)
  (ii) distribution (microscopic)
  (iii) incidence

(b) Congestion:-
  (i) extent
  (ii) incidence

Fatty Change:-

In 5,8 percent of cases, fatty change was diagnosed macroscopically (27 percent of these were described as mild, 26 percent as moderate and 4,5 percent as severe), 37 percent as normal and 3,7 percent as pale. When present the fatty change was centrilobular in 33% of cases reviewed histologically.

Congestion:-

In 55 percent of cases, congestion was noted at necropsy and categorised as mild (30%), moderate (23%) and severe (2,2%). The liver was described as normal (non-congested) in 38%. Histologically hepatic congestion was noted in fifty percent of cases. It was of acute onset, mild to moderate in extent and centrilobular in distribution.

Hepatic Necrosis:-

The incidence of hepatic necrosis was 13 percent, with varying degree, in the cases examined, histologically; sixty percent of these cases with necrosis displayed focal non-specific necrosis, consistent with shock or sepsis; in one third of cases centrilobular necrosis was present and in seven percent with massive centrilobular necrosis.

5.5 THE SPLEEN

A plasma cell immune response is a consistent proven finding, which is an indication of active antibody formation in the spleen (8). This response may be slight or marked. Previously, Sevitt had interpreted splenic eosinopaenia as synonymous with adrenal hyperactivity (1). However, Teplitz was unable to correlate this finding with any definite clinical or pathological finding and hence is not regarded as of any morphological usefulness in this later series (8).

Diffuse congestion of the spleen is the commonest finding in burns victims; cellular infiltration monocytes and phagocytosis were rarely seen, in a recent publication (9).

5.5.1 Findings: The Spleen

The spleen was enlarged (weight for age) in 24,4 percent, softened in 33,5 percent and congested in 47 percent of cases with macroscopic lymphoid hyperplasia in 13 percent of cases.
Microscopically congested splenic sinusoids were present in all cases reviewed. Acute splenitis was not seen as frequently as expected, as the softening at post-mortem could have resulted due to autolysis and also the prolonged post-mortem interval. This has been the experience of other authors (8).

5.6 THE GASTROINTESTINAL TRACT (GIT)

Complications involving the GIT may occur in the early or late phases after sustaining burns. In general, the larger the burn and the older the patient, the higher the incidence. Some complications may be transient while other may be life-threatening.

5.6.1 Complications involving the Gastrointestinal Tract

5.6.1.1 Acute Gastric and Duodenal Ulcers ("Curling's" Ulcer)

Curling, in 1842 described the occurrence of duodenal ulcers in burn victims. These ulcers may occur in the stomach or duodenum and are indistinguishable from the acute stress ulcers occurring in other pathological conditions.

5.6.1.1.1 Definition

Erosions are lesions confined to the superficial layers of the gastric or duodenal mucosa, but if the full mucosal thickness or muscularis mucosae is breached, the term acute gastric or stress ulcer is applied (8).

5.6.1.1.2 Incidence

The incidence of acute ulcers varies between twenty-five and forty-seven percent (8) (10), with a peak incidence within the first seventy-two hours; other authors found a peak incidence after seven to ten days (16). In the series by Teplitz, the incidence was double that of retrospective routine autopsy analyses, since both gastric erosions and ulceration were included. Acute stress ulcers were previously associated with a concurrent bacteraemia (10). The decreasing incidence of Curling's ulcers is associated with the following factors:-

i) the control and reduced frequency of major septic complications;
ii) introduction of prophylactic antacids via the nasogastric tube, H_2 receptor anatgonist and intermittent nasogastric suction;
iii) improved nutritional supplementation of the victim.

Despite the advances in the management of burns patients, GIT haemorrhage remains a potentially fatal complication with a mortality of 0,16%. The incidence of GIT ulceration has decreased over the last twenty years to between one and five percent of burns patients with the use of prophylactics (19).

5.6.1.1.3 Occurrence

Acute stress ulcers may occur in burn patients with twenty to thirty percent of the body surface area.
All age groups are afflicted with a slight female preponderance in one series (8).

5.6.1.1.4 Aetiology and Pathogenesis

The aetiology and pathogenesis of the lesion is poorly understood and thought to be multi-factorial, involving complex mechanisms, with many influences. The factors involved are: (7) (10) (16)

i) increased acid secretion with back diffusion;
ii) decreased production of bicarbonate buffer;
iii) reduction of regional blood supply;
iv) accumulation of acid ions;
v) damage to the mucosa and decreased production of mucus;
vi) bacteremia and major septic complications;
vii) local mesenteric thrombosis;
viii) mucosal infarction;
ix) negative nitrogen balance with depression of epithelial regeneration;
x) therapy (aspirin, steroids and anti-inflammatories)

5.6.1.1.5 Morphology

Stress, alcohol, smoking steroid or anti-inflammatory induced ulcers are similar in appearance. Refer to Table 5.3 for a tabulated contrast of stress and peptic ulcers.

TABLE 5.3 - CONTRASTS IN STRESS AND PEPTIC ULCERATION

	STRESS	PEPTIC
Sex	Slight female predominance	Male predominance
Age	All ages (9 months to five years) 43%	Older age group
Site	Fundus - posterior - 80%	Pylorus - anterior location
Number	Usually multiple (78%)	Usually solitary
Size	Gastric ulcer is smaller than duodenal ulcer - usually 0,5 - 1,0cm	Gastric ulcer - smaller Duodenal ulcer - larger
Configuration and Shape	Poorly defined and circular	Greater depth, round or oval
Appearance	No induration, oedema or injection Minimal inflammation. No fibrosis	Indurated or punched out, oedema with chronic inflammation. Heals by fibrosis. Mucosal overhang with puckering
Base	Dark brown stain due to blood and acid digestion. No fibrosis or scarring	Slough present Fibrosis and scarring. Smooth and clean due to peptic digestion of exudate

5.6.1.1.6 Complication of Gastroduodenal Ulceration

i) Fatal haemorrhage

Acute stress ulcers in the duodenum are more likely to give rise to fatal haemorrhage.

ii) Perforation

Perforation usually occurs in anterior ulcers with development of secondary peritonitis; the defect may be sealed off by fibrinous exudate and covered by omentum.

5.6.1.1.7 Findings: Stress Ulceration

In this series, the criteria used for the diagnosis of stress ulceration was the macroscopic presence of ulceration at post-mortem. In addition the site was recorded (oesophagus to large bowel), associated haemorrhage and time interval after burn injury sought. Examination of all the hospitalised victims case sheets showed that the clinical diagnosis was not made in any of the cases. In all cases, the diagnosis was made at necropsy being 7,5% (10 patients) of hospitalised patients. The commonest site in this series was the oesophagus (5 cases), followed by the fundus (3 cases) and duodenum in 2 cases. Neither haemorrhage nor perforation of the gastrointestinal tract was evident. No evidence of acute colonic ulceration or perforation was reported in any of the cases in this series (of one hundred and fifty one).

5.6.1.2 Acute Gastric Dilatation

Acute gastric dilatation may occur as a complication of tube feeding, septicaemia, oxygen therapy via a nasal catheter and aerophagia. It has a peak occurrence around the seventh day.

The effects of this condition are acute gaseous or fluid distension of the stomach, regurgitation with subsequent dehydration and electrolyte imbalance; untreated it leads to circulatory collapse (10) (13).

The cause of acute gastric dilatation is unknown; factors that may play a role in its development are reflex inhibition of gastric motility, gastric atony and aerophagia due to poor epiglottic control (8).

5.6.1.2.1 Findings:

In this series, no evidence of acute gastric dilatation was recorded in clinical records or at post-mortem. Case sheets of eighty-six hospitalised patients were examined and in the remaining cases, a written clinical summary of the deceased sojourn as an in-patient, was relied upon.

5.6.1.3 Paralytic Ileus

This condition occurs more frequently in those cases with greater than forty-five percent burns of total body surface area. It is caused by acute gastric ulceration, septicaemia especially with staphylococcaemia and prolonged immobilization with constipation (16).

5.6.1.3.1 Findings:

In this series, no clinical or pathological evidence of paralytic ileus was present. Case sheets and summaries were studies as outlined above.

5.6.1.4 Colonic Ulceration

Recently, acute colonic ulceration with severe burns have been reported, especially when episodes of hypotension had occurred. Penetration of the colonic wall up to the serosa have been described but no perforations have been observed (2). None were noted in this series.

5.6.1.5 Findings:

The incidence of gastrointestinal tract complications was 7,5 percent in this series, when compared with other studies (26). As noted earlier, complications are proportional to the age of the patient, extent of burns and the rate of sepsis. The mean age in this series was twenty-six years, probable accounting for the low incidence of ulceration.

5.7 ADRENAL GLANDS

Consequent to the stress of sustaining burns, urinary and blood steroid levels are elevated. This increased functional activity correlates with a functional hypertrophy of the glands. Lipids depletion is a common finding, as is cortical and medullary sinusoidal congestion.

Infrequently massive, bilateral haemorrhage is found at necropsy with fibrin thrombi in the sinusoids. With a prolonged post-mortem interval, autolysis combined with congestion, may simulate haemorrhage. Haemorrhage often correlates with fulminant Gram negative septicaemia. Sevitt's interpretation of splenic eosinopaenia as a manifestation of adrenal activity, has not been reproduced by other researchers (1) (8).

5.7.1. Findings:

Adrenal haemorrhage in 3 percent (4 cases) of hospitalised patients was recorded, in this series.

The adrenal glands were lipid depleted in 46,5 percent of hospital cases or 43 percent of all fatal burns.

Microscopically, cortico-medullary junction congestion aside no other pathology was detected.

5.8 THE CENTRAL NERVOUS SYSTEM

The effects of thermal injury to the brain are mainly secondary in nature (to other occurrences), e.g. raised intracranial pressure and its sequele and secondary to water and electrolyte imbalance (6) (14).

Histologically, no pathognomonic changes are apparent to the neurons. However, degenerative changes may be present in the cerebellar Purkinje cells, as in hypoxia or carbon monoxide poisoning.

An entity termed "Burn Encephalopathy", or neurological syndrome is seen in children with burns and scalds (14). The symptoms and signs are cerebral irritability, high fever, vomiting, twitching, convulsions and coma, with an occasional fatality. The neurological disturbance is postulated to be due to cerebral dysfunction secondary to water and electrolyte imbalance.

When dehydration is treated too aggressively via rapid infusion of a large volume of water intravenously, the body fluids revert from a state of hyperosmolality to hypo-osmolality. A "lag" develops at the blood-brain barrier, the cerebro-spinal fluid (CSF), which is hyper-osmolar, is subjected to an osmotic equilibration, causing water to enter the CSF. Cerebral oedema results, leading to hypothalamic mal-function.

The "body thermostat" is reset at a higher level, to the point where shivering occurs, while peripherally vasoconstriction persists to minimize heat loss. Hence, the core of the body or core temperature is hyperpyrexic but the "outer shell" temperature remains below normal (14).

5.9 HAEMATOPOETIC RESPONSE

Following severe thermal injury, several haematopoetic changes occur. Granulocytosis, thrombocytopaenia, megakaryocytosis and anaemia develop.

Anaemia is a common occurrence. Besides haemorrhage and haemolysis of red cells, research has demonstrated that the red cell production rate is depressed (12). The anaemia that occurs in burns victims can be classified into:

a) early (hours)
b) later (days), and
c) delayed (weeks) - following the injury (12).

5.9.1 The Multifactorial Aetiology of the Anaemia in Burns

5.9.1.1 Early changes

Haemorrhages and haemolysis occur during the initial post-burn period at the burn site. This manifests as anaemia in burned patients within hours of injury. The haemolysed cells are the source of the haemoglobinaemia and haemoglobinuria. The presence of free circulating haemoglobin in the plasma of a patient with recent burns is indicative of severe injury (12).

5.9.1.2 Later changes

i) Haemolysis of heat-damaged cells from the early phase;
ii) destruction of red cells as they circulate through the burned area;
iii) haemorrhage at the time of change of dressing and debridement;
iv) thrombosis in the capillaries of the burned area (8);
v) depressed rate of erythropoeisis.

5.9.1.3 Delayed changes

i) Further haemorrhage during surgically related procedures;
ii) from gastrointestinal haemorrhage, as described in 5.6;
iii) depressed rate of erythropoeisis.

Overwhelming sepsis and fat embolism may induce anaemia in patients with burns (10).

Thrombocytopaenia is a frequent finding in patients with burns, and has been used to predict mortality; patients with thrombocytopaenia have a higher mortality rate than those who maintain a peripheral blood platelet count within a normal range (12). Although a megakaryocytosis is evident, the precise mechanism inducing the thrombocytopaenia has not been fully explained. It would appear that thrombocytopaenia is the result of the peripheral consumption of platelets rather than marrow failure (12).

Erythroblastopaenia was described previously (32) and recently confirmed (12). The latter researchers found morphological evidence of reduced erythropoeisis, i.e. reduced amounts of erythroid tissue in the bone marrow of patients with fatal burns, and a lack of extramedullary haemopoeisis. They postulated that an, as yet unidentified, inhibitory substance may be responsible for the suppression of erythroid tissue, resulting in anaemia.

5.9.2 Leucopaenia

Luecopaenia, as well as impaired phagocytosis, is an early feature seen in severe burns. Sequestration and destruction of white cells in burned tissue and bone marrow depression by chemical factors, released from damaged and necrotic tissue, jointly aggravate the leucopaenia. Excessive fluid therapy, part of the anti-shock treatment in burns, leads to granulocyte trapping in tissue ravaged by burns (20).

In a series of twenty-two fatalities, post-burn granulopoeisis had apparently increased when compared with septic, non-burned patients, implying that an appropriate increase in white cell production does occur in fatal burns (12).

5.9.3. Immunological function

After a burn injury, both the humoral and cellular immune functions are depressed, altering all immune functions and reducing host resistance. The incidence and extent of the immunlogical disruption are proportional to the burn size and are greatest in patients who sustain burns of greater than fifty percent of total body surface area. The suppressed immunity predisposes the victim to burn wound sepsis, and infection elsewhere in the body. The patient's resistance to opportunistic bacterial, fungal or viral infections is diminished or lost (17).

5.9.3.1 B-Lymphocytic Activity

The effects of the B-lymphocytic activity depression are:-

a) suppresion of the reticulo-endothelial system,

b) low levels of immunoglobulins, due to decreased synthesis and loss into the burn exudate.

Protein depletion and impaired immunoglobulin synthesis, predispose the burn victim to bacterial infection.

5.9.3.2 T-Cell Activity

A depression of T-cell activity, after severe burns, impairs resistance to bacterial, viral and fungal infection. The source of infection may be endogenous, from the burn wound contaminants, or exogenous, from the nasal passage and hands of medical and paramedical staff (20).

5.9.3.3 Haematological Abnormalities

The spectrum of haematological abnormalities in the local cases, were restricted to 86 cases admitted to King Edward VIII Hospital.

(i) Haemoglobin

(a) Decreased (below normal reference range) 27%

(b) Elevated 5,8%

(c) Normal 30,2%

(d) No Record 37,7%

(ii) White Cell Count

(a)	Elevated	43%
(b)	Decreased	10,46%
(c)	Normal	6,98%
(d)	No Record	39,5%

(iii) Platelets

(a)	Normal	15,1%
(b)	Decreased	6,98%
(c)	Increased	5,80%
(d)	No Record	39,5%

5.10 SEPTICAEMIA

Septicaemia by definition is a clinical state with bacteremia accompanied by clinical symptoms and signs (chills, fever, rashes and petechial haemorrhages - the latter two are rarely seen in burns or at necropsy). It is indicative of inadequate/overwhelmed/failed host resistance to localise the infection. The organism may enter the blood by direct extension into a vessel, as is possible in burns by mycotic emboli, infected lymphatics and subsequent access to the venous system. Signs of fatality are oliguria, jaundice and intravascular coagulation. The common causative agents are beta-haemolytic streptococci, staphylococci, Escherichia coli and other gram-negative coliform bacteria and Pseudomonas. Macroscopically splenomegaly, hepatomegaly and lymphadenopathy with focal necrosis, multiple petechial haemorrhage due to capilliary damage, adrenal haemorrhage and icterus may be present, not necessary simultaneously. At post-mortem specific damage is difficult to detect. The acute septic spleen is enlarged, mushy or diffluent, with naked eye appearance of hyperplasia. The softness of the spleen may also be due to autolysis, which in addition alters the microscopic findings significantly and irreversibly.

Histologically, the splenic germinal centres are hyperplastic, large and poorly defined; the reticular elements are hyperplastic and the sinusoids show erytrocyte and polymorphonuclear leucocytes. In the presence of bacteria foci of acute inflammation too may be present. In fulminant septicaemia death soon ensues within hours and few if any pathological changes are present at post-mortem (33, 34).

The incidence of septicaemia using the above criteria was investigated in eighty six hospitalised patients of variable duration of stay in hospital. Although an elevated white cell count was recorded in 43% of these cases, only 3% positive blood cultures were found. The prolonged post-mortem interval in this series, did not assist either. Hence, pathologically no definite diagnosis of septiacemia was possible.

5.11 FINDING: CHEMICAL PATHOLOGY

Biochecimal Parameters.

The biochemical derangements in this series were as follows:-

A	SODIUM	%
(1)	Hyponatremia	19,77
(2)	Hypernatremia	4,65
(3)	Normal	43,02
(4)	No Record	36,04

B	POTASSIUM	%
(1)	Hyperkalemia	19,74
(2)	Hypokalemia	4,65
(3)	Normal	40,69
(4)	No Record	34,88

C ALBUMIN

Of 86 cases examined only fourteen levels of albumin were recorded and ranged from low to very below normal.

5.12 FINDINGS: MICROBIOLOGY

The local cases admitted to King Edward VIII Hospital survived on the average for 7 days, ranging from admission to 18 days. Burns patients when able were transferred to Clairwood Hospital and then if demise are referred for a medico-legal necropsy.

In the 86 admitted patients, burn wound swab for culture of organisms was taken in 25% of admissions (22 patients). No documented record of the number of blood culture was available, while only three positive blood cultures were recorded.

The common causative organism in available burn wound sepsis, are as follows:-

	%	NO.
(1) Staphylococcus	40,9	9
(2) Pseudomonas	36,36	8
(3) E. Coli	31,82	7
(4) Klebsiella	22,72	5
(5) Streptococcus	9,09	2
(6) Proteus	9,09	2

In 87% of burn wounds more than one organism was cultured.
The positive blood cultures results were as follows:-
(1) Staphylococcus
(2) Pseudomonas
(3) Klebsiella and Proteus

Hence, the common source of infection in burns, is the colonisation of burn wounds.

5.13 SUMMARY

In this study, the following complication causing death were encountered (TABLE 5.4):

TABLE 5:4

CAUSES OF DEATH FOLLOWING BURN INJURY

1. Bronchopneumonia	39%
2. Shock	5%
3. Acute Tubular Necrosis	17%
4. Inhalational Injury	14%
5. Pulmonary Embolism	4%
6. Liver Necrosis	1%
7. Extensive Charring	1%
8. Pulmonary Complication(Bronchopneumonia and Pulmonary embolism)	43%

Pulmonary complication was the most common complication, accounting for forty three percent of the mortality. This findings is in keeping

with studies by other authors (26) (27) (28).

The high incidence of sepsis in burn victims may be due to the lack of a burn unit in the greater Durban area; burns patients are treated in general surgical or paediatric surgical wards, with a high rate of cross infection (Professor R Mickel, personal communication).

The mortality due to post-burn shock remains high, as shown in this series and elsewhere (26) (27) (28). Since patiens are referred to hospital sooner and shock more adequately treated, bronchopneumonia presents as the main cause of death, albeit delayed.

CHAPTER 5

REFERENCES

1. Tedeschi C.G, Ackroyd F.W, Eckert W.G and Tedeschi L.G. ed. Forensic Medicine, Volume 1, Mechanical Trauma, 1st edition. Philadelphia: W.B. Saunders, 1977: 715-728.
2. Pruitt B.A and Goodwin C.W. Burns. In: David C Sabiston, ed. Textbook of Surgery 12th edition Philadelphia: W.B. Saunders, 1981: 287-316.
3. Aikwana N. et al. Multiple organ failure in burned patients. Burns, 1987; 13 (2): 103-109.
4. Robbins S.L, Cotran R, Kumar V. Pathological Basis of Disease. 3rd edition. Philadelphia: W.B. Saunders, 1984: 114, 1026-1035.
5. Herndon D. et al. Respiratory injury, Part 1. Burn Care and Rehabilitation, 1986; 7 (2): 184-195.
6. Schiavon M. et al. Renal Damage in severely burned patients. Burns, 1988; 14 (2): 107-114.
7. Anderson J.R. The Urinary System. In: J.R. Anderson, ed. Muir's Textbook of Pathology. 11th edition. London: Edward Arnold, 1985: 138-145.
8. Teplitz C. The Pathology of Burns. Artz C.P. and Moncrief J.A, ed. The Treatment of Burns. 2nd edition. Philadelphia: W.B. Saunders, 1969: 22-28.
9. Chen Yi-Sheng, Davies J.W.L. et al. Histopathological and ultra-structural changes in liver tissue from burned patients. Burns 1985; 11 (6): 408-418.
10. Curreri P.W. Burns. In: Schwartz S.I. Principles of Surgery: 3rd edition. New York Mc Graw Hill: 286-301.
11. Wartman W.B. Mechanisms of Death in Severe Burn Injury. In: C.P. Artz, eg. Research in Burns. 9th edition. Washington: American Institute of Biological Sciences, 1962: 6-14.
12. Wallner S.F. and Warren G.H. Haematopoietic response to burning. Burns, 1985; 12 (1): 22-27.
13. Pruitt B.A, and Goodwin C.W. Burns. In: David C. Sabiston, ed. Textbook of Surgery 12th edition. Philadelphia: W.B. Saunders, 1981: 287-316.
14. Settle J.A.D. Burns, the first forty-eight hours. Hertfordshire: Smith and Nephew Pharmaceuticals, 1974: 23-24.

15. Bloch Cecil. Burns Handbook. Pinetown: Smith and Nephew Medical Division, 1987: 7-33.

16. Hummel R.P. Clinical Burn Therapy. 1st edition. Massachusetts: John Wright, 1982: 411-428.

17. Scotti T.M. and Hackel D.B. Heart. In: Kissane J.M. ed. Anderson's Pathology. 8th edition. S.E. Louis; C.V. Mosby Co. 1985: 630-631.

18. Chen Yi-Sheng, Davies J.W.L. et al. Histopathological and ultra-structural changes in liver tissue from burned patients. Burns, 1985, 11(6): 408-418.

19. Prasad J.K., Thomson P.D. and Feller I. Gastrointestinal haemorrhage in burn patients. Burns, 1987; 13(3): 194-197.

20. Mickel R., Lloyd D, et al. Burns Resuscitation Manual Chapter None. Pinetown: Natal Blood Transfusion Service: 1-18.

21. Schwär T.G., Loubser J.D., Olivier J.A. Exposure to Temperature Change. In: The Forensic ABC in Medical Practice. 1st edition. Pretoria: Haum Educational Publishers, 1988: 198-215.

22. Kinsella J. Smoke Inhalations. Burns, 1988; 14(4): 269-279.

23. Prien T. and Traber D.L. Toxic Compounds and inhalational injury - a review. Burns, 1988; 14(6): 451-460.

24. Miyoshi K., Tsukada S., Yasuda Y. et al. Hepatic disorder in burn patients. Burn, 1985; 12(1): 49-53.

25. Davies M.R.Q., Cywes S., Davies D. A Review of Deaths in a Paediatric Burns Unit. South African Medical Journal, 1976; 50: 1479-1483.

26. Haberal M., Oner Z., Bayraktar U., Bilgin N. Epidemiology of Burns in Turkey. Burns, 1987; 13(2): 136-140.

27. Abu Ragheb Sameeh, Qaryoute Salah and Husam El-Muhtaseb. Mortality of Burns in Jordan. Burns, 1987; 10(6): 439-443.

28. Bang R.L. and K.M. Mosbah. Epidemiology of Burns in Kuwait. Burns, 1988; 14(3): 188-193.

29. Stone H, Martin J.D, Jr.: Pulmonary injury associated with Thermal Burns. Surgery Gynecology Obstetrics 1969; 129: 1242-1246.

30. Foley F.D, Moncrief J.A, Mason A.D. Pathology of the lung in fatally burned patients. Annals of Surgery 1968; 167: 251-264.

31. Shook C.D, Macmillan B.G, Altemeir W.A. Pulmonary complications of the burn patient. Arch Surg 1967; 97: 215-224.

32. Lelkes G and Bernat I. Electron microscopical study of the bone marrow of burned patients. Haematologia 1970; 4: 295.

33. Walter J. and Israel, M.S. General Pathology 5th edition Edinburgh; Churchill Livingstone 1979; 100-101.

34. Peery T.M and Miller F.N. Pathology 2nd edition Boston, Little, Brown and Co. 1971; 89-9

CHAPTER 6

SPECIAL TYPES OF BURNS

6.1 ELECTRICAL BURNS

6.1.1 Introduction

Despite the widespread use of electricity, death to to electrocution is comparatively rare, although the utilizers of this energy source possess limited or no knowledge of electrical current.

6.1.2 Aetiology

Most causes of electrical injuries are accidental, while deaths of a suicidal, homicidal and judicial nature are rare. (Refer to TABLE 6.1).

TABLE 6.1 - THE AETIOLOGY OF ELECTRICAL BURNS

CAUSES	EXAMPLES
1. Accidental	i) **Occupational:** electricians, construction workers, crop sprayers, etc. ii) **Home Accidents:** faulty appliances, ignorance, children climbing into transformer compounds, etc. Use of electrical appliances in the bathroom.
2. Homicidal	Use of electric batons and small amperage current during torture.
3. Suicidal	Rarely seen.
4. Judicial	Execution by the use of the electric chair, primarily in the U.S.A.

6.1.3 Pathophysiology of Electrical Burns (1)

The effect of electricity on the human body depends on the following factors:

6.1.3.1 Closure of electrical circuit

Closure of electrical current: the passage of current between parts of the body interposed between two conductors possessing different electrical potential.

6.1.3.2 Alternating vs direct current

a) Humans are four times more susceptible to alternating current than direct current;
b) The greater the frequency the less the injury - the dangerous range is between 39-150 cycles per second.

6.1.3.3 Voltage/electrical potential

Low voltage current is more dangerous than high voltage (greater than 1000 Volts) current. Voltage is dangerous because:

a) only a brief duration of exposure to current is required;
b) of the development of electrical arc or flame;
c) it crosses the skin barrier more easily and frequently (3).

6.1.3.4 Amperage or current strength

Amperage is the most important factor in electrocution because of the "hold on" phenomenon at 15 m Amp.

6.1.3.5 Resistance of the body

The resistance of the various tissues is inversely proportional to the water content, that is, the higher the water content the lower the resistance, i.e. the skin offers high resistance while blood and other body fluids offer low resistance. For current to flow through the body:

a) the resistance of the skin has to be overcome, by wetting or charring;
b) the body needs to be earthed.

As the skin resistance is overcome, by moisture or burning, the amperage through the skin and tissue rises and the electrical current is converted to heat or thermal energy. Sweat is produced, which in turn further decreases the resistance. Heat production in the tissue rises precipitously (Joule's Law: Power or Heat $P = I^2R$, where I is the current flow and R is the resistance), producing the so-called "internal burn" which is produced by conversion of water into steam (3).

6.1.3.6 The Path of the Electrical current

An electrical current passing through the body, follows the shortest route or the pathway of least resistance through the point of entry and exit. The magnitude of the current is maximal along this route. Vulnerable organs, such as the heart and brainstem, are easily overcome, if along the pathway (2).

6.1.3.7 Duration of the contact

The greater the duration of contact, the greater the magnitude of current and hence the greater the injury or effect (1).

6.1.3.8 Pre-existing Disease

In individuals with cardiac disease, there is a rapid development of ventricular fibrillation when accidentally exposed to electricity (3).

6.1.4 Pathology of Electrical Burns

6.1.4.1 Low Voltage Current (Less than 1000 Volts)

6.1.4.1.1 Specific Injuries

6.1.4.1.1.1 The Skin

Absent injury or burns by contact, electrothermal burns (flash or arc burns), flame burns or metallisation may be present.

i) Burns by Contact

These include the entry and exit wounds. The injury at the entry wound is usually more extensive than at the exit site and is usually second to third degree burns.

ii) Electrothermal Burns (Flash arc burns)

These burns are usually encountered with very high voltage burn (2500-40 000. V), where heat is generated external to the skin. The burn is sustained without contact. These burns are deep in extent and severe in consequence.

iii) Flame Burns

This injury is described when the clothing is ignited by electrical sparks and arcing. In electrical injuries, the physiological response to the trauma is grossly disproportionate to the magnitude of surface area involvement unless there is coexistent considerable secondary thermal injury. In addition, clinically profound acidosis, oliguria and myoglobinuria are a frequent occurrence, which would lead to tubular plugging and acute renal failure (3).

iv) Macroscopic appearance of Electrical Injury

a) Superficial Wounds

Superficial wounds are caused by electricity involving the epidermis. These wounds are grey-white in colour with a parched appearance (1).

b) Deep Wounds

These wounds induced by electricity resemble a crater, with a rolled edge and surrounding hyperaemia and bullae (1).

v) Histological Appearance of Electrical Injury

Microscopically, in the epidermis, splits or bullae develop. In the basal layer, cells and nuclei are elongated, hence the description of the palisade arrangement. The dermis is acellular, loosened from the deeper layers, and the collagen is denatured.

Electron microscopically, a loss of the dermazone layers, degeneration of cells and nuclear membranes, mitochondrial destruction and a release of the basal membrane are apparent (1).

6.1.4.1.2 Non Specific Injuries

6.1.4.1.2.1 Muscle and Tendon

The electrothermal sequele of injuries to muscle is directly or indirectly due to vascular changes. In the presence of infection, septicaemia may develop. Extensive cellular trauma to muscles can be a source of myoglobin, leading to acute tubular necrosis (1).

6.1.4.1.2.2 Blood Vessels

Blood acts as a conductor when a current passes through it and thus larger vessels are more effective dissipators of heat and are therefore less prone to injury than smaller vessels. The heat is injurious to the endothelium, resulting in micro-embolisation and vascular thrombosis with partial or total occlusion of the lumen, resulting in muscle death. Oedema occurs in the subcutaneous and deep tissue due to the vascular changes (1).

6.1.4.1.2.3 Bones and Joints

Severe muscle contractions lead to fractures and dislocation of joints. Hairline fractures occur due to heat.

6.1.4.1.2.4 The Cardiovascular System

In delayed deaths, non-specific findings such as coronary artery spasms, diffuse end-arteritis and diffuse myocardial injury may contribute to death.

6.1.4.1.2.5 The Nervous System

Loss of consciousness is common, lasting for seconds to hours, while coma and death do result. Survivors complain of a multitude of symptoms, ranging from amnesia, headache, drowsiness, lethargy and disturbed respiratory rhythm.

With immediate death, no macroscopic or microscopic changes are evident, as expected. When delayed deaths occur, petechial haemorrhage, focal degeneration of ganglionic cells, demyelination and fragmentation of neuronal axons and perivascular necrosis are observed. The aetiology of the injury is not directly linked to the electrothermal injury, but due to anoxia or hypoxia. Associated head injury may cause extradural, subdural, subarachnoid, intracerebral and intraventricular haemorrhages. Damage to the lens of the eye has also been recorded (1) (3).

6.1.4.1.2.6 The Lungs

Secondary changes resulting in pulmonary oedema, intra-alveolar and subpleural petechial haemorrhages are occasionally found. Infection may be present. Alveolar epithelium swells and desquamates (1).

6.1.4.1.2.7 The Kidneys

Secondary changes in the kidney are usually present and are of multifactorial aetiology, that is, primary shock, direct injury to renal epithelium and glomeruli, breakdown of protein products and myoglobinuria, leading to acute tubular necrosis (1) (3).

6.1.4.1.2.8 The Pregnant Uterus

Foetal death and precipitous delivery may occur (2).

6.1.4.1.2.9 The Gastrointestinal Tract

i) Colonic perforations are the most common site of injury.
ii) Haemorrhage and/or necrosis in the ileum, oesophagus, liver, gall bladder and pancreas may be seen (1).

6.1.4.2 High Voltage

This entity is described as voltage greater than 1000 Volts.

Depending on the period between shock and death, a multitude of injuries may be responsible for death, for example, cardiac arrhythymia, myocardial infarct, shock lung, acute renal failure, liver failure, amongst others (3).

6.1.4.3 Lightning

Lightning is the discharge of an electrical field which has emanated from the clouds or the negative static or electrical field of discharge in the clouds.

At the time of the discharge, the potential difference (or voltage) between the earth and the clouds is 10^8 Volts. The strength of the current in lightning nears 10,000 Amp.

In North America, most lightning injuries occur between May and September, while locally, between September and March. A single stroke of lightning lasts for one hundredth of a second.

Persons most prone to lightning injuries are those individuals who, during a thunderstorm, seek shelter below a tree, near a flagpole, or in a boat on water (1).

6.1.4.4 Findings

In the present series 3,9% of patients died due to electrical injuries. All these were accidental in nature, where the entrance wound was usually in the upper limb. The victims died at the place of injury (at work) and were not hospitalised.

No cases of electrothermal and flame burns related to electrical injuries were present in this series.

6.2 MECHANISM OF DEATH IN ELECTRICAL INJURY

Death occurs due to one of the following:

6.2.1 Cardiovascular System

Terminally one of the following may occur:

a) ventricular fibrillation (most common),
b) cardiac arrhythymia,
c) cardiac arrest.

Persons with pre-existing cardiac or coronary artery disease, are more prone to these complications (2).

6.2.2 The Respiratory System

One or more of the following may precede death:

a) Paralysis of the respiratory centre in the brainstem with consequent respiratory failure and apnoea occurs. This takes place if the electrical current follows a path through the brainstem.
b) Spasm of the respiratory muscles, for example, the intercostals and diaphragm with apnoea and hypoxia, occurs.
c) Tetanic Asphyxia
This occurs due to spasm and hypoxia of the respiratory muscles and the heart.
d) Burns sustained due to exposure to high voltage current
Death may occur in the absence of passage of current through the person's body.
e) Injuries induced by electrical shock, for example, fall from height.
f) Secondary Delayed Complications
i) infected necrotic skin, subcutaneous tissue, muscle;
ii) haemorrhage;
iii) acute tubular necrosis;
iv) multi-organ failure. (1)

6.2.3 Findings

No fatality due to lightning injury was noted for the year sampled.

6.3 CHEMICAL BURNS

Although rare, this type of burn can lead to severe problems. Most lethal chemical burns are caused accidentally, at home or in industry, homicidally or in military conflict.

6.3.1 Pathophysiology

In burns sustained due to chemicals, the offending agent is the chemical rather than heat. Usually contact with the skin is for a longer duration, be it on the skin, within the skin or below blisters. The injury is hence a progressive one. The extent of damage on tissue is influenced by:

i) the concentration of the chemical;
ii) mode of skin contact;
iii) degree of penetration;
iv) specific mechanism of action for that chemical;
v) duration of contact. (3)

Skin exposed to chemicals is destroyed by:

a) coagulation necrosis,
b) vascular thrombosis, with prolonged exposure,
c) denaturation of protein, especially collagen, which undergoes denaturation and precipitation due to oxidation and reduction ("corrosion"),
d) cellular dehydration, intercellular oedema of the integument and subcutaneous tissue, and separation of the dermis and epidermis may occur.

With the disruption of the keratin layer, the deeper lying dermis is more extensively damaged. Strong alkalis and acids lead to extraction of water from tissue, and protein is precipitated with loose alkali-albuminate bond development, which is water soluble. Other agents inhibit cellular metabolism, as they are protoplasmic poisons (4).

The mechanism of injury is a combination of chemical and thermal (exothermic) effect on tissue. (Refer to TABLE 6.2)

Phosphorous is an exception since it produces thermal burns in air at room temperature.

TABLE 6.2 - HISTOLOGICAL FEATURES OF ACID AND ALKALI BURNS

HISTOLOGY - ACID AND ALKALI BURNS
Epidermis may appear intact but is not.
Cytoplasmic oedema.
Epidermal perinuclear vacuolization with elongated pyknotic nuclei
Dermal - epidermal separation.
Collagen banding and loss of collagen
Intercellular oedema with separation and irregularity of cells of sebaceous and sweat glands
Endothelial thickening and thrombosis of deep dermal capillaries

The sequele of chemical burns are due mainly to scarring, for example, blindness, strictures, contractures and hepatotoxicity (2).

CHAPTER 6

REFERENCES

1. Schwär T.G, Loubser J.D, Olivier J.A. Exposure to Electricity and Ionising Radiation. In: The Forensic ABC in Medical Practice. 1st ed. Pretoria: Haum Educational Publishers, 1988: 217-228.
2. Artz C.P. and Moncrief J.A. Special Types of Burns. In: The Treatment of Burns 2nd ed. Philadelphia W.B. Saunders, 1969: 214-230.
3. Tyler T.G. Treatment of Special Burns. In: R.P. Hummel, ed. Clinical Burn Therapy, 1st ed. Massachusetts: John Wright Publishers, 1982: 193-238.
4. Van Rensburg L.C. An Experimental Study of Chemical Burns, South African Medical Journal, 1962; 36: 754-759.

CHAPTER 7

THE MORTALITY OF BURNS

In most developing and some developed countries, a paucity of literature and statistics on the true mortality of burns exists. Greater attention is paid to the high incidence of death caused by motor vehicle collisions, which supercedes burn fatalities (1)(3)(4). Most burns are accidental and the high risk, burn-prone individuals have been identified as children below six, and the aged.

Prior to 1900, almost all patients with third degree burns, or second degree burns greater than fifteen percent of total body surface area, died. Between 1900 and 1960, gradual improvement in the treatment and mortality supervened. The advent of topical antibiotics in 1967, further reduced the mortality. In hospitalised burn patients fatality is influenced by the occurrence of complications, especially infection; this aggravates the burn victim's condition. Presently, seventy-five percent of patients with burns greater than thirty percent of total body surface area, succumb to the effects of infection - a high and unacceptable rate (2). In the United Kingdom, there has been a steady decline in mortality, primarily due to the decrease in the number of deaths in children and young adults, and the better control of septicaemia and burn wound sepsis with topical antibiotics, a "burns team" approach and successful campaigning for the prevention of burns (6). The average mortality for burns in developing countries in 1967 was 2,91 per 100 000, while in 1972 the mortality had marginally decreased to 2,64 per 100 000; in 1977 it was 2,66 per 100 000. This fluctuating rate was not uniform for all countries.(1)

Where conditions are near optimum, the mortality is low, e.g. in Holland. The incidence of burns in the Netherlands is five thousand per year (0,03% of the population), of whom five hundred die (0,125% of burned patients, or 0,004% of the population). Seventy percent of all burns victims are children between three and twelve, while the elderly outnumber the industrial burn victims. All burns admissions and deaths in Holland are documented for epidemiological analysis. Associated organisations educate scholars, workers and the adult population at large on burns prevention (5).

In contrast, in the United States of America, four thousand children die annually due to burns, being surpassed only by death due to motor vehicle accidents (4).

7.1 FACTORS INFLUENCING THE MORTALITY OF BURNS

Many factors influence the mortality of burns, the most important being the following:

i) extent of burn;
ii) age of victim;
iii) depth of burn;
iv) region of body burnt;
v) interval between burn and anti-shock therapy initiation;
vi) concomitant injury;
vii) health and tolerance of victim;
viii) alcohol and drug abuse;
ix) epilepsy.

7.2 MORTALITY IN DURBAN AND DISTRICTS

Local fatal burn statistics for 1988 are as follows:

TABLE 7:1

1.	Durban Metropolitan Area	160 deaths
2.	Chatsworth Mortuary	50 deaths
	Dr D Pillay - personal communication	
3.	Verulam Mortuary	36 deaths
	Dr TG Govender - personal communication	
		246 deaths

Addington Hospital treats twenty burn victims, mainly Whites and Coloureds, annually; King Edward Hospital admits four hundred burn patients annually primarily Black and Indian children; several other Indian children are admitted to the R.K. Khan Hospital. Of the 110 deaths in the paediatric surgical wards in 1988, twenty-nine patients or twenty-six percent died as a result of burns. Homes fuelled on products other than electricity, emerged as the common denomination (Professor R. Mickel, personal communication).

7.3 CONCLUSION

The high morbidity and mortality of burn injuries needs to be reduced by firstly instituting preventative measures, secondly anticipating and treating the complication and thirdly to treat infections effectively. More effective topical antibiotics, the aggressive management of septic complications, the establishment of well equipped burns units and professionally trained personnel are highly recommended to achieve this reduction (3).

Economically, burns remain one of the more expensive conditions to treat, in view of the protracted stay in hospital and the provision of a large burns medical personnel. In addition, regular changes of special dressings, antibiotics and multiple transfusions of blood products, all add to the escalating cost of management.

CHAPTER 7

REFERENCES

1. Chaurasia A.R. Mortality from burns in developing countries. Burns, 1983; 9(3): 184-185.
2. Sameeh Abu Ragheb et al. Mortality of burns injuries in Jordan. Burns, 1984; 10(6): 439-443.
3. Davies M.R.Q. et al. A Review of Death in a Pediatric Burns Unit. South African Medical Journal, 1976; 50: 1479-1487.
4. MacMillan B.G. and Friedberg D.L. Special Problems of the Pediatric Burn Patient. In: Hummel R.P. ed. Clinical Burn Therapy 1st ed. Massachusetts: John Wright and Sons, 1982: 429-431.
5. Gunay M. Burns Prevention in Europe. Burns, 1983; 9(5): 315-317.
6. R.F. Brown. Injury by Burning. In: J.K. Mason ed. The Pathology of Violent Injury 1st ed. London: Edward Arnold Publishers Ltd., 1978: 113-136.

PERSONAL COMMUNICATIONS

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CHAPTER 8

THE PREVENTION OF BURNS

8.1 INTRODUCTION

The prevention of burns is dependant on epidemiological studies, relevant to the specific area sampled. The approach to the prevention of burn injury has to be tailored for each geographic region and varying socio-economic groups. The modes of prevention instituted need to be reviewed regularly and altered when necessary. Variations in burn studies are best deduced from systematic epidemiological surveys. Manifestation of the efficacy of preventative measures take a decade or two to become obvious and present as a decrease in the incidence, morbidity and mortality of burns.

8.2 BURNS PREVENTION PROGRAMME

The campaign for burns prevention is based on the following scheme:

i) identification of the cause(s);
ii) education;
iii) legislation.

8.2.1 Identification of the Cause(s) of Burns

This may be a single source, a wide spectrum of sources, or a way of life or habit. Single source aetiology such as faulty appliances or inflammable nightwear can be modified, or banned by legislation. In developing countries, where incomes are low, it is difficult to enforce legislation.

Many burns arise from mishaps in the home for example, due to floor level open fires, during cooking or heating of liquids, lit cigarette stubs, playing with matches, falling into fires, candles falling onto beds. The victims of these are usually children (1).

Women are prone to burns in the kitchen, where loose-fitting flammable clothing (for example saris, nightdresses or nylon clothing) catch fire easily; incorrect and negligent use of Primus pressure stoves and gas cylinders lead to explosions (1).

Accidents at work usually occur due to leakage of steam and chemicals, improper electrical wiring and spillage of hot liquids such as tar and molten metal. Faulty handling of equipment, fatigue, carelessness, lack of information, attentiveness and understanding, and being under the influence of alcohol contribute to the occurrence of burns (1).

Burn accidents in industry are governed by safety regulations and laws with a measure of accountability while similar constraint on domestic mishaps is absent. Hence there is a high incidence of accidental burns in the home (over 80%) in developing countries.

8.2.2. Education

Arousing awareness of the dangers and prevention of burns should be aimed at as large a segment of the community as possible by such means as the press, radio, television, school programmes and organisations, burn associations and scout movements (2).

The target in burns education should be children, as they tend to be more open-minded, logical, receptive and experiment with new ideas and accept change more readily. Training and education in the safety, prevention and first-aid of burns should be instituted as early as the pre-school level and pursued with persistence and precision throughout the schooling career. Education in the safe use of matches, Primus pressure stoves, gas and electrical appliances and the danger of fireworks should be stressed (2).

Women engaged in household chores should be advised to wear tightfitting cotton, woollen or other garments which are more resistant to ignition (1).

The older generation are more resistant to change but the provision of bathing aids, for example non-slip bathmats, grabrails or bath seats make bathing safer in this high risk group. In addition, safer water-heating appliances (geysers against immersion heaters or open unguarded fires) protect against scalds in the aged and young (4).

Individuals should be taught the first-aid treatment of burns as it is invaluable in minimising the extent of burn injury. This entails cooling the burned area with cold water. Older people are sceptical of this approach and need to be convinced in adopting this method (1).

Burns must be part of the medical curriculum during training of medical personnel and should be classified a "social disease". Burns prevention should be taught to nurses, medical students and doctors, in detail, including the diagnosis and treatment of burns (1).

National burns associations should liaise with consumer movements regularly with the aim of creating greater awareness, improving product quality and the level of service (1).

8.2.3 Legislation

As yet, legislation specifically aimed at burn prevention does not exist in South Africa. There is a need to promulgate laws aimed at improving the safety of potentially hazardous products. In Denmark appropriate legislation against unsafe fireworks, flammable clothing and toys have led to a decrease in the incidence of burns (3). In the United Kingdom and Holland, similar legislation has been equally effective.

8.3 RECOMMENDATIONS

Most burns are accidental and therefore preventable. In the United States of America 75% of fires and 85% of burn injuries are preventable (2). The aim of prevention is to decrease the number of accidental injuries due to carelessness or ignorance, rather than treat extensive burns in esoteric, well-equipped burns units.

The prevention of burns is more economical than any palliative or curative measures undertaken.

Burns-related injuries are preventable as is evident in Holland, Denmark and the United Kingdom, where recent studies show a decreased incidence. The above countries have instituted an active Burn Prevention Programme, as outlined earlier (3).

When compared with the rest of the world, burns programmes in South Africa are still in their infancy. Educational syllabuses and legislation need to be initiated, to counter the ravages of burn injuries. The less privileged, illiterate majority of the population should be the target group. The problem is complex, and the solution involves a politico-socio-economic strategy, involving the state, provincial and local health agencies, medical personnel, fire departments, social workers, education authorities, associated burns organisations and parental guidance. Regional burns associations, including the Lions and Rotary clubs can be of great assistance in the school education programmes.

In rural areas, an educational campaign of burns prevention, including first-aid, through Primary Health Care Centres is a dire necessity. This function can be ably performed by a trained nurse or social worker, or other suitably qualified individual, well versed in the official and native languages of the population (1).

Visual aids, using slides, videos and posters, can be used to good effect, especially in regions of high illiteracy. Slides on television and in cinemas and regular broadcasts on radio have also proven very effective. Television, being a very powerful medium of communication, can be used very effectively, influencing a large audience of different age groups simultaneously. The feasibility and benefit of these types of plans have paid dividends when it was instituted in Bombay, India (1).

The author strongly recommends that burns be classified as a registerable or notifiable "social" disease. This would greatly improve the availability of epidemiological data presently deficient, but essential in planning prevention programmes.

At the tertiary education level medical students should be trained and adequately tutored in the prevention, first-aid management and pathology of burns. This knowledge can then be disseminated when the newly qualified practitioner sets up practice and passes on the information to patients, their families and friends, associates and peers.