HYPERTENSION IN BURNED CHILDREN

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TABLE OF CONTENTS

	Page
ACKNOWLEDGEMENTS	iii
TABLE OF CONTENTS	iv
LIST OF TABLES	vi
Chapter	
I. INTRODUCTION	1
Statement of the Problem	2
Purposes	2
Background and Significance	2
Definition of Terms	6
Limitations	7
Delimitations	8
Assumptions	8
Summary	8
II. REVIEW OF LITERATURE	10
Introduction	10
Hypertension Associated with Poliomyelitis	20
Incidence of Hypertension in Burned Children	24
Hypertension Associated with Resuscitation	25
Hypertension Associated with Cardio-Vascular Failure	28
Hypertension Associated with Electrolyte Imbalances	30
Sodium	30
Potassium	34
Calcium	37
Hypertension Associated with Renal Disease	40
Hypertension Associated with Hematologic Changes	46
Hypertension Associated with Elevated Renin Levels	53
Hypertension Associated with an Atypical Form of Disseminated Intravascular Coagulation	54
Hypertension Associated with the use of Hexachlorophine	59
Hypertension Associated with Psychological Factors	61
Summary	65

Page

III. PROCEDURE FOR COLLECTION AND TREATMENT OF DATA 66
Setting 66
Population 67
Tool 68
Data Collection 69
Treatment of Data 70
Summary 71

IV. ANALYSIS OF DATA 72
Analysis of Data 72
Summary of Findings 86
Summary 90

V. SUMMARY, CONCLUSIONS, IMPLICATIONS AND RECOMMENDATIONS 91
Summary 91
Conclusions 95
Implications 97
Recommendations 98

APPENDIX 101

BIBLIOGRAPHY 107

LIST OF TABLES

		Page
1.	Early Mortality and Total Mortality in Various Size Burns According to Age	11
2.	Mortality	12
3.	Admissions by Month Over Ten Year Period	73
4.	Admissions by Year Over Ten Year Period	74
5.	Admissions by Age	75
6.	Admissions by Sex and Race	76
7.	Patients by Size of Burn	77
8.	Admissions by Type of Burn	78
9.	Admissions by Family History of Seizures or High Blood Pressue	79
10.	Adequacy of Resuscitation	80
11.	Hypertension Associated with the Use of Phisohex	81
12.	Relationship Between use of Phisohex and Type of Burn Injury in Hypertensive Patients	82
13.	Number of Days Phisohex Baths were Given	82
14.	Occurrence of Sepsis	83
15.	Postburn Day of Sepsis	83
16.	Family Members Involved in the Burn-Related Accident	84
17.	Patients Classified as Battered Children	84
18.	Patients with Pulmonary Injury	85
19.	Survival Rates	85

CHAPTER I

INTRODUCTION

Hypertension as a complicating factor in the disease process often occurs in conditions involving the kidneys, circulatory, or central nervous system. However, hypertension is noted to occur in two instances where no clinical reason can be stated: namely, in patients with poliomyelitis and in pediatric burn patients.

Although the published material is scanty, the incidence of this hypertension is frequent enough that more information is needed. By documentation of the frequency and a description of the high-risk population, it is possible that prevention of an unsuspected, progressive hypertension resulting in seizures may result. The critical care nurse is in a position to recognize these high-risk patients if sufficient information is available. In addition, the nurse must understand the related nursing role in the early detection of the hypertensive pediatric burn patient. Thus, the importance of recognizing the pediatric burn patient who is a high-risk candidate for this, as yet, unexplained hypertension has great significance in the role of the critical care nurse in the prevention and treatment of hypertensive complications.

Statement of the Problem

The problem of this study was to investigate the incidence of hypertension in the pediatric burn patient.

Purposes

The purposes of the study were to:

1. Determine the incidence of hypertension in the pediatric burn population.
2. Determine the cause of hypertension in the pediatric burn patient.
3. Describe the pediatric burn population with hypertension.

Background and Significance

Significant arterial hypertension, often heralded by the abrupt occurrence of convulsions, frequently develops in children with burns of more than 30 percent total body surface area but in relatively few adults (Baxter 1967:919). Although the incidence of hypertension is recognized, a description of the high-risk population is not available. For this reason, the assessment of the pediatric burn patient with hypertension requires further investigation. The background and significance of this study describes the history and significance of hypertension in patients with poliomyelitis and pediatric patients with burns. Included is the role of the critical care nurse regarding these patients.

Because of the small numbers of published reports of patients with burn hypertension, a survey of the hypertension associated with poliomyelitis, in the absence of clinical cause, is indicated. Both types of patients appear to develop a significant level of hypertension in the absence of any clinical etiology. The similarities of the two disease courses may give some clues as to the etiology of the hypertension.

Gurlee and Panos (1948:24), in an epidemic of poliomyelitis in Minneapolis in 1948, observed transient hypertension in the first week of the disease in 72 percent of seventy cases of bulbar poliomyelitis. McDowell and Plum (1951:241) published their observations on ninety-five patients with poliomyelitis of whom 47 percent developed hypertension. Mechelka and Linke (Lackmund 1950:450) observed 114 patients, 54 percent of whom developed a transitory hypertension.

Blood pressure readings were often not obtained due to the large number of patients being treated during these years of epidemic poliomyelitis and to the difficulty of obtaining blood pressure readings of patients in iron lungs; nevertheless, the incidence of elevated blood pressure in the absence of any abnormal clinical symptoms was noted. The incidence was generally estimated at between 50 and 70 percent of the reported cases.

The etiology of the blood pressure elevations was presented in different classifications in many of the studies. Some investigators reported that its development in most cases was related to hypoxia

(Weinstein and Shelokov 1951:281) or to hypercapnia (Sack and Bernsmeier 1950:886). Others concluded that since the incidence of hypertension was not related to the localization of paralysis, emotional factors, anoxia, sympathetic system involvement, or kidney involvement, it must be inferred that it was probably of central origin (Perlstein et al. 1953:628; Lachumnd 1950:450). Kemp (1957:109) raised the possibility that ganglionic cell degeneration in the nucleus magnocellular in the medulla oblongata was a necessary condition for the development of hypertension. With the development of the poliomyelitis vaccine and the subsequent decline in the number of poliomyelitis patients, the exact cause of the hypertension associated with poliomyelitis was never proven conclusively.

At about this same time, hypertension of unknown etiology in pediatric burn patients was first described. In 1944, Gibson and Brown (1944:49) described burn children with mental symptoms such as drowsiness, delirium, and at times, convulsions resulting from hypertension. Morrison (1947:129) concluded that the main cause of illness and death in the early stage of the burn course, when seizures tend to occur, was the reduction of blood volume by plasma loss without adequate replacement. He also stated that hemodilution may occur when fluid replacement is too vigorous. It seems, however, that hemodilution occurs rarely as a possible contributing factor to seizures following burns, but may be an important factor in patients with sudden hyponatremia (Hughes and Cayaffa 1973:203). However, even after the recognition of the importance of adequate

resuscitation, the hypertension persisted in a significant number of pediatric burn patients.

Lowrey (1967:140) had a major role in describing hypertension in children with burns by showing a higher mortality rate in these children than in the normotensive group. In fifty-three children, 24 percent demonstrated sustained hypertension. However, Antoon et al. (1972:6) failed to find a single case of burn encephalopathy based on hypertension. Hughes et al. (1973:347) stated that the incidence of seizures in the 662 children studied from 1950-1971 was 5.4 percent. In addition to these population surveys, many individual case histories of hypertensive burn children were found in the literature (Berliner, Shenker, and Weinstock 1972:92; Joshi 1970:2130; Yost and Holmes 1974:1147).

The data thus far has reflected the varied findings and the difficulty encountered in attempting to describe the population of hypertensive burn children. To be able to give competent care, the critical care nurse must understand the pathophysiology involved. The nurse may be able to rely on previous experiences in giving care to these children, but assessment would be much more valuable if additional parameters were made available. Nurses provide most of the child's care; their contact is closer and more continuous than that of any other individual, and they function as intermediaries between the child and his environment, whether it be medical or familial (Quinby and Bernstein 1971:90). Because of close contact

with the pediatric burn patient, the nurse is in a position to recognize the child whose burns place the child in a high-risk population for the development of hypertension and to initiate appropriate nursing intervention.

Definition of Terms

For the purpose of this study, the following definitions were used:

1. Burn - the destruction of tissue due to any thermal, chemical, or electrical injury.
2. Hypertension - the normal diastolic blood pressure by age as described plus 20 mm Hg which persists for at least twenty-four hours (See Appendix A).
3. Child - any person 13 years of age or younger.
4. Normal Laboratory Values - those values which enable the cell to maintain normal functioning through a situation of dynamic equilibrium (Reed and Sheppard 1971:4). Normal laboratory values as established by Parkland Memorial Hospital and used in this study were those of sodium, potassium, serum calcium, blood urea nitrogen, serum creatinine, hemoglobin, hematocrit, white blood cell count, and platelet count (See Appendix B).
5. Hyperthermia - a rectal temperature of greater than 101° F.
6. Hypothermia - a rectal temperature of less than 98° F.
7. Parkland Formula of Resuscitation - Lactated Ringer's solution given intravenously according to the following:

4 cc lactated Ringer's/kg body weight/percent total body surface area burn with total being given in the first 24 hours postburn. (Baxter et al. 1974:131).

8. Burn Sepsis - When the patient is normovolemic, the clinical picture may include (1) hypotension; (2) high cardiac output; (3) normal or increased blood volume; (4) normal or high central venous pressure; (5) low peripheral resistance; (6) warm, dry extremities; (7) hyperventilation; and (8) respiratory alkalosis. When the patient is hypovolemic, the clinical picture may include (1) hypotension; (2) low cardiac output; (3) high peripheral resistance; (4) low central venous pressure; and (5) cold, cyanotic extremities. Development of mild hyperventilation, respiratory alkalosis and an altered sensorium may be the earliest signs of gram-negative infection (Shires, Carrico, and Canizaro 1973:154).

Limitations

The recognized limitations of this study were:

1. The researcher had no control over the completeness or accuracy of the data which was recorded on the chart.
2. The patient and/or his family were not available for follow-up studies.

Delimitations

The delimitations of this study were:

1. The population consisted of the children who were admitted to Parkland Memorial Hospital with a primary diagnosis of burns between June 1, 1965 and June 1, 1975.
2. The children had no pre-existing renal, cardiovascular, or central nervous system diseases.
3. Demographic data was collected on all of the children who met the above criteria; in addition, vital signs and laboratory value studies were collected on all hypertensive children.

Assumptions

The assumptions for this study were:

1. The burn process alters the body functions.
2. Children respond to the burn process in a different manner than adults respond.

Summary

Significant arterial hypertension frequently develops in children with burns. Although the incidence of hypertension is recognized, a description of the high-risk population is not available For this reason, the assessment of the pediatric burn patient with hypertension requires further investigation.

Chapter II presents available information on the suggested causes and frequency of the hypertension which occurs in the pediatric burn population. Since hypertension in the absence of clinical explanation also occurred in patients with poliomyelitis, a review of the literature on hypertension associated with poliomyelitis is included.

Chapter III discusses the procedure for the collection and treatment of data obtained from the charts. Included is the development of the tool for data collection and its modification for use by the department of biostatistics.

Chapter IV presents the analysis of data of the demographic study of the entire population of children with burns. Further anlysis of the clinical data from the charts of children in the population who developed hypertension is included.

Chapter V includes a summary of the data and conclusions derived from the computerized analysis of the data. Conclusions are drawn from data statistically significant. Implications and recommendations based on the data are made.

CHAPTER II

REVIEW OF LITERATURE

Introduction

Each year at least 2 million persons are burned seriously enough to require medical attention or to restrict their activity for a day or more. Of these, about 100,000 require hospitalization and from 9,000 to 12,000 die (Beland and Passos 1975:116).

Burns outnumber all other causes of death during infancy, childhood, and adolescence with the highest incidence occurring in children under 5 years of age. The most frequent causes of burns in children are burns from hot water which occur when (1) the child is left unsupervised in the tub and turns on the hot water tap, (2) the child is placed in a tub of hot water that has not been tested, or (3) hot liquid is spilled on the child. Burns from flames, electrical burns, caustic acid or alkali burns, chemical burns, and smoke inhalation injuries to the respiratory tract also occur in children. Burns inflicted upon the child as a result of child abuse are also seen (Brunner and Suddarth 1974:1256).

In a report on 412 pediatric burn patients treated at Brooke Army Medical Center from 1959 through 1968, the cause of the burn was as follows: 303 patients from flame burns; 93 patients from scald burns; and 16 patients from contact burns. Playing with matches, open space heaters and stoves, and accidents involving gasoline or kerosene

constituted the cause of the bulk of the flame burns. Scald burns were caused most commonly by the child pulling containers of hot liquids down upon himself or turning on the hot tap in an unattended bathtub. A few burns were caused by direct contact with a stove or a hot iron (Bruck, Asch, and Pruitt 1970:658).

Mortality, in general, increases in proportion to the size of the burn. The one variable which does not relate to the size of the burn is the added component of an inhalation injury. In a study of the mortality rate of 388 pediatric burn patients resuscitated by the Parkland formula (4 cc Ringer's lactate per kilogram body weight/percent of burn in the first 24 hours) Baxter, Marvin, and Curreri (1973:707) presented data on early mortality (first 10 days) and total mortality in various size burns according to age as follows:

TABLE I

EARLY MORTALITY AND TOTAL MORTALITY IN VARIOUS SIZE BURNS ACCORDING TO AGE

AGE		Less Than 20%	20-39%	40-59%	60-79%	Greater Than 80%	Totals
0 to 2	TP*	110	32	6	–	2	150
	EM+	–	9.4	–	–	100	3.3
	TM++	–	9.4	10.6	–	100	4.0
3 to 7	TP	87	38	16	4	3	148
	EM	–	–	6.2	25	–	1.3
	TM	–	–	25.0	100	100	7.4
8 to 15	TP	48	27	6	6	3	90
	EM	–	–	–	16.6	–	1.1
	TM	–	–	–	33.3	66	4.4

*TP – Total Patients
+EM – Early Mortality
++TM – Total Mortality

SOURCE: Baxter, C.R.; Marvin, J.A.; and Curreri, P.W. "Fluid and Electrolyte Therapy of Burn Shock," Heart and Lung Journal 2 (1973):707.

Thus, in this group of patients, the mortality rate for children under 8 years of age with burns greater than 60 percent total body surface area was 100 percent. This figure decreased markedly in patients in the 8 to 15 year age group.

In a study of 412 pediatric burn patients, Bruck, Asch, and Pruitt (1970:658) presented analysis of mortality according to percent body surface burn which included the component of pre- and post- Sulfamylon use.

TABLE 2

MORTALITY

Per Cent Total Body Surface Burn

	0-30	30-40	40-50	50-60	60-70	70-80	80-100	Totals
Group I*								
Deaths/No. Pts	4/92	16/22	18/23	13/17	10/11	10/10	10/10	81/185
Per Cent Mortality	4.5	73.7	78.3	76.5	90.9	100	100	43.8
Group II**								
Deaths/No. Pts.	0/139	5/32	6/20	5/15	11/14	3/4	3/3	33/227
Per Cent Mortality	0	15.6	30.0	33.3	78.6	75.0	100	14.5

*Conventional therapy, 1959-1963
**Sulfamylon topical therapy, 1964-1968

SOURCE: Bruck, Harold; Asch, Morris; and Pruitt, Basil. "Burns in Children - A Ten Year Experience with 412 Patients," Journal of Trauma 10 (1970):658.

The overall mortality in patients receiving the conventional therapy was 43.8 percent as compared to 14.5 percent in patients receiving Sulfamylon therapy. The difference in mortality was most striking in the 30 to 40 percent group, and was impressive in the 40 to 60 percent groups. There was some slight improvement in the 60 to 70 percent group.

The care of the burned patient is a unique, challenging, and rewarding experience for those who have chosen to enter this field of medicine. The multiplicity of care requirements during the course of medical management and patient recovery necessitates the use of the special skills and knowledge of the nurse, physical therapist, occupational therapist, dietitian, social worker, chaplain, and a host of other allied medical personnel in a team approach to the total care of the patient. The nurse is one of the key members of the burn care team. The ability to work efficiently with others, a basic understanding of the physiology and pathology of the burn injury in general, and the burn wound in particular, are necessary. The nurse is responsible for the provision of a clean and safe environment for the patient and monitoring of procedures for prevention of cross-contamination. As the individual in around-the-clock attendance, the nurse is in a unique position to provide close scrutiny of the wound. The patient's general physical and mental status must be evaluated continually with early signs of potential problems identified and reported to appropriate team members by the nurse (Hunt, McGranahan, and Pruitt 1973: 690).

The general basic principles of burn care for adults apply to children, but there are some anatomic, physiologic, and psychologic differences that modify a child's reaction to the burn injury. The child below the age of 2 years has a relatively higher mortality

rate in burns of over 20 percent than other age groups. The child below the age of 2 years has immature kidneys. Because of glomerulotubular immaturity, the infant kidney is unable to excrete sodium, chloride, and some other ions. It cannot reabsorb water readily, so large volumes of hypertonic urine are produced. The immature kidney cannot readily excrete large volumes of nonelectrolyte fluid; therefore, while it is very easy for an infant to become dehydrated, he can also become overhydrated. Infants and children have a larger body surface area in proportion to their weight than do adults, increasing their potential for water evaporative loss. In addition, peripheral circulation is labile in the infant. The myocardium functions well, but the the peripheral compensation is poor. Pneumonia, atelectasis, and constricting chest eschars are a real danger to the pulmonary system of the younger child. While the gas and exchange ventilation is well stabilized shortly after birth, the infant's high metabolic rate, coupled with the stress of the burn trauma, leaves very little marginal reserve. The older a child is, the greater his margin of safety (Jacoby 1972:94).

The nursing management of pediatric burn patients is complicated, to say the least. Attempts to deliver comprehensive care to these patients outside of a structured burn unit is next to impossible. Yet in 1973, Feller and Archambeault (1973:10) reported less than 100 of the 6,000 general hospitals in the United States provided specialized burn care. The primary purpose of the burn treatment

facility is to provide the resources required for care of burn accident victims. The authors also state that a special hospital facility is of little value without adequately skilled medical personnel to use it. Only 41 of the 92 medical schools in the United States studied were affiliated with hospitals offering any specialized burn care, and only 9 of these had Burn Centers for teaching and research as well as for patient care. The net effect of this shortage of medical skills and facilities for burn treatment is that approximately 90 percent of all burn patients do not receive the quality of care they need. All patients with burns serious enough to require hospitalization require some level of specialized treatment. In the United States, at this time, there is no complete training program in burn care for physicians and only two programs offering a master of science degree in nursing care of the thermally injured exist.

Feller and Archambeault (1973:32) state that during the lengthy process of wound healing, many potentially lethal complications can occur. These complications are similar to those which can beset any critically ill patient, and their treatment is similar. The unique fact about burn patient care is that life-threatening complications are the rule rather than the exception.

Schwartz et al. (1974:268) report that the victim of thermal injury often is beset by various complications, singly or in combination. While most of these occur in the first two weeks postburn, they also frequently occur several weeks after the initial injury.

A composite list of complications which may occur at the time of or after the burn injury encompasses all body systems. The cardiovascular system response may include vascular thrombosis secondary to the burn, hypovolemia or hypervolemia, hemodilution or hemoconcentration, increased viscosity, vasoconstriction or vasodilation, arterial thrombosis, right-or-left-sided heart failure, cardiac arrythmias, visceral necrosis secondary to low flow states, disseminated intravascular coagulation, and cardiac failure secondary to a circulating myocardial depressant factor. Pulmonary system response may include respiratory acidosis or alkalosis, impaired pulmonary gas exchange producing hypoxia and/or hypercapnia, pulmonary embolism, pneumonia, respiratory distress syndrome, pulmonary edema, obstruction of the upper airway secondary to edema, and bronchospasm secondary to smoke inhalation.

The metabolic system may respond by producing metabolic acidosis or alkalosis, electrolyte imbalances, hormonal imbalances, hypothermia or hyperthermia, and catabolic metabolism secondary to starvation. Renal system response may include oliguria, dysuria, high output renal failure, myoglobinuria, and renal artery spasm. Other systems affected may include the hepatic system resulting in liver failure and the gastrointestinal system resulting in bleeding or Curling's ulcers. The central nervous system response may include disorientation, seizures, coma, and responses secondary to the failure of other systems.

Iatrogenic complications may result from the use of various drugs and anesthetic agents, ventilators, central venous and arterial lines, urinary catheters, bathing techniques, and surgical complications.

The body's response to the burn injury is formation of scar tissue and contractures. When not managed adequately, physical deformity is another complication. Sensory deprivation and psychological trauma are an expected complication of the hospital course of the burn patient. Added to the possible complications of all burn patients is one which appears to occur mainly in children; namely, hypertension of unknown etiology.

In a specialty area such as the burn unit, there is a constant need for skilled nursing care, and the nursing staff must assume unique responsibilities (Feller and Archambeault 1973:373). If the patient is not critically ill, the nature of his condition is often such that he may become critically ill at any time. The nurse must be able to recognize medical emergencies and to institute proper corrective measures immediately; permission is given to perform specific medical tasks and to make decisions which ordinarily would be made by doctors. The nurse working in a burn unit therefore has duties which cannot be carried out without adequate training and education.

Few other patients are as ill or require such skilled care. The burned individual is undergoing the ramifications of the most severe form of trauma and the nurse's knowledge base must be extensive in order to make the frequent necessary alterations in patient care. Education beyond the depth and scope usually encountered is needed to prepare practitioners capable of bringing this expertise to the bedside. For this reason, the role of the clinical nurse specialist, prepared at the master's level, is indicated.

Kenner and Marvin (1975:22) state that the burn clinical nurse specialist should be able to administer high-quality patient care including assessment, planning, implementation, and evaluation, and be capable of meaningful collaboration with other health disciplines. The specialist should be an effective teacher both in educational and clinical settings, and should be a role model while participating in patient care. The specialist should be innovative, willing to seek help from the literature and other appropriate sources, and capable of planning and participating in research studies. The clinical nurse specialist is the person responsible for the standards of burn care nursing administered on the burn unit and, as such, develops a well-planned orientation and continuing education plan. The clinical nurse specialist is thus in a position to assure that the burn nurse is able to fulfill the potential of the role as described by Feller and Archambeault (1973:379):

1. The nurse is expected to 'observe and report.' To do this, she must know what signs and symptoms are pertinent and must be reported, and to whom to report them. As she becomes more sophisticated, she knows what corrective steps will be taken and has the needed supplies and equipment on hand.
2. She is expected to 'follow the doctor's orders.' The doctor's orders can be viewed as the plan of action. It is frequently assumed the nurse will know the reason for an order, correctly implement it, and be aware of what situation would contra-indicate the order.
3. There are many situations in nursing where specific orders are never written. These include care relating to general comfort, hygiene, infection control, emotional support, and most emergency situations. In these, the nurse is expected to take independent action and seek assistance as it is needed. Nursing care orders are a guide for these unwritten orders.

4. The nurse is given multiple assignments of patients with different needs, and must be able to place priorities on her activities to adequately and effectively meet these patients' needs.
5. Each procedure the nurse carries out has been formulated as a solution to a particular problem. The procedure is the plan and the act. If the nurse doesn't understand the importance of the procedure when she is setting her priorities, she may view it only as a mechanical act and may not carry it out with a definite objective in mind, or she may not adapt it to fit the needs of the individual.

When the nurse has the proper background to recognize the pathophysiologic changes which accompany the burn injury, it is possible to determine objectives in patient care. Brunner and Suddarth (1975:1258) state that the general objectives of nursing care of children with burns are:

A. To recognize the symptoms of shock and to know support measures which are initiated to restore and maintain circulation.
B. To observe for symptoms of respiratory distress and initiate measures to alleviate distress.
C. To provide scrupulous skin care in order to prevent infection and promote healing.
D. To provide a high protein, high calorie diet in order to provide nutrition necessary for healing and for the growth and development needs of the child.
E. To maintain a planned physical therapy program in order to achieve the greatest functional capacity for the child.
F. To prepare the child for the many painful and surgical procedures he must undergo.
G. To provide emotional support for the child who has been very frightened and traumatized by this painful experience.
H. To support the parents during this very difficult time.

A careful evaluation of each patient and his needs is essential to the delivery of nursing care to the burned child. Although hypertension is one of many complications which may occur, the physical and mental damage which it can precipitate makes it a

very serious complication. The nurse must be aware of the many factors which may precipitate a hypertensive episode in the pediatric burned patient and, if possible, act to halt or correct the factors.

Hypertension Associated with Poliomyelitis

Hypertension as a complicating factor in the disease process often occurs in conditions involving the kidneys, circulatory, or central nervous system, However, hypertension is noted to occur in two instances where no clinical reason can be stated: namely, in patients with poliomyelitis and in pediatric burn patients. Since both types of patients appear to develop a signficant level of hypertension in the absence of any specific clinical etiology, it was felt that the similarities of the two disease courses might give some clues as to the etiology of the hypertension.

Gurlee and Panos (1948:24), in an epidemic of poliomyelitis in Minneapolis in 1948, observed transient hypertension in the first week of the disease which persisted for an average of 3.4 days in 72 percent of 70 cases of bulbar poliomyelitis. McDowell and Plum (1951:241) reported an abnormally elevated arterial blood pressure in over half of the 103 patients with acute paralytic poliomyelitis seen at the New York Hospital during a two year period. The hypertension appeared in the acute stage of the disease and sometimes lasted well into convalescence; its degree was, in general, proportionate to the severity of the patient's illness. Arterial

hypertension, with a diastolic blood pressure greater than 90, appeared in 45 of 95 adolescents and adults with acute anterior poliomyelitis. Males were twice as frequently affected with hypertension as were females. The highest incidence of hypertension occurred in patients suffering bulbospinal paralysis or paralysis of all four extremities; 29 of the 30 patients developed arterial hypertension. Anoxia, hypercapnia, or artificial respiration appeared to intensify and prolong the otherwise transient hypertensive state that occurred.

Mechelka and Linke (Lachmund 1950:450) observed 114 patients with poliomyelitis 54 percent of whom developed a transitory hypertension. Lachmund (1950:450) studied 208 patients, 10 percent of whom developed hypertension. After exclusion of every other possible genesis, this increase in blood pressure was regarded as centrally caused. In all of the autopsied cases, a lesion in the medulla oblongata was found. Sack and Bernsmeier (1950:886) opposed the idea of a central origin of hypertension. They felt that the blood pressure increases in disease of the central nervous sytem did not always follow the same course. In 4 cases which they studied, the hypertension was felt to be due to hypercapnia.

Perlstein et al. (1953:628) noted hypertension as a frequent occurrence in 195 poliomyelitis patients treated in Chicago in 1950. They reported that hypertension occurred in about one-third of all cases and that it was generally transitory in nature, although

in two instances, it was prolonged. Hypertension was three times more frequent in patients with paralysis than in non-paralytic patients, and it was most common in respiratory cases. This study reported the incidence of hypertension was not related to the localization of paralysis, emotional factors, anoxia, sympathetic system involvement or kidney involvement; therefore, it was inferred that the hypertension was probably of central origin.

Kemp (1957:109) studied 427 patients admittted to a hospital in Copenhagen in 1952. Some patients incurred acute kidney lesions which probably caused hypertension. Other patients, however, who apparently were well-ventilated and without kidney lesions, developed hypertension which occasionally persisted in spite of marked clinical shock symptoms. Hypertension was found to be equally frequent in children and adults, males and females. Hypertension persisting for days was found during the acute stage in about 55 percent of the patients who died, 30 percent of the bulbar cases, 45 percent of the bulbospinal cases, and 40 percent of the very severe spinal cases. Neither artificial ventilation per se, nor hypo- or hyper-ventilation seemed a necessary factor or sufficient cause for the development of hypertension; nor did azotemia, changes in the spinal medulla, in the diencephalon, or in the telencephalon. It was felt that ganglionic cell degeneration in the nucleus magnocellular in the medulla oblongata was a necessary condition for the development of hypertension.

Weinstein and Shelokov (1951:281) reported hypertension as relatively common in acute poliomyelitis. They felt its development was related to hypoxia; return to the normotensive state was produced by maintaining adequate ventilation.

Sennett et al. (1951:529) reported a case of a 16 year old white male who developed a case of sustained hypertension complicating acute poliomyelitis with a protracted course of 24 weeks, the longest reported period of survival of a patient still in an acute phase of the disease. It was felt, in this case, that the hypertension probably was due to neurogenic factors.

Steigman et al. (1952:264) studied 226 patients seen in a 1950 epidemic. Severity of poliomyelitis was related to frequency of hypertension; it was found in 120 of 186 paralytic patients, but in only 5 of 40 non-paralytic patients. Age was not an important factor. There was hypertension in fifty of eighty-two paralytics 0 to 4 years old; twenty-nine of forty-four paralytics 5 to 9 years; seventeen of twenty-six paralytics 10 to 14 years; and twenty-four of thirty-four paralytics 15 to 35 years. Hypertension appeared early in the illness and usually was under 14 days duration.

Blood pressure readings were often not done due to the large number of patients being treated during these years of epidemic poliomyelitis and to the difficulty of obtaining blood pressure readings of patients in iron lungs. However, the incidence of elevated blood pressure, in the absence of any abnormal clinical symptoms, was generally estimated at between 50 and 70 percent of the reported cases.

The etiology of the blood pressure elevations was presented in different classifications in many of the studies. Some investigators reported its development in most cases as related to hypoxia (Weinstein and Shelokov 1951:281) or to hypercapnia (Sack and Bernsmeier 1950:886). Others concluded that since the incidence of hypertension was not related to the localization of paralysis, emotional factors, anoxia, sympathetic system involvement, or kidney involvement, it must be inferred that it was probably of central origin (Perlstein et al. 1953:628; Lachmund 1950:450). Kemp (1957:109) raised the possibility that ganglionic cell degeneration in the nucleus magnocellular in the medulla oblongata is a necessary condition for the development of hypertension. Arfonad was the drug which was reported to be the most effective in controlling the acute hypertension of poliomyelitis (Steigman 1954:343).

With the development of the poliomyelitis vaccine, there was a subsequent decline in the number of poliomyelitis patients. Thefore, the exact cause of the hypertension associated with poliomyelitis was never proven conclusively.

Incidence of Hypertension in Burned Children

Hypertension is reported as a complication in the pediatric burn population. The incidence varies greatly but its occurrence is well documented. Clinical investigators (Lowrey 1967:140) reported that in a population of 53 burned children, 24 percent were hypertensive, with diastolic pressures in excess of 90 mm. Hg. The hypertension appeared usually within the first two weeks after the burn

but could occur at any time during the first two months. All age groups were involved, with blood pressures ranging as high as 230/160. Duration of symptoms was usually short but did persist from several weeks to 2 months. In addition, the mortality rate was definitely higher in the hypertensive children. Baxter (19673:915) states that significant arterial hypertension develops in 20 percent of children with burns of more than 30 percent total body surface area. Seligman, Carroll, and MacMillan (1971:655) reported on 7 cases admitted to their institution with 3 of the 7 developing hypertension of unknown etiology. Hughes et al. (1973:347) report seizures in 5.4 percent of 662 children, with hypertension as a factor in some of the cases. Reported cases of hypertension with an apparent clinical cause, i.e., hypernatremia, renal failure, etc., also appear in the literature.

Hypertension Associated with Resuscitation

When a patient is burned, one of the immediate results is a loss of the integrity of the skin as it functions as a water barrier. This conversion of skin to a freely water-permeable state initiates a series of changes in body composition that can rapidly endanger life, and even prove fatal if not treated adequately (Davies and Liljedahl 1970:59). In addition to fluid lost through the burn wound itself, fluid is sequestered in the intracellular space in the burned and in the unburned areas of the skin. In burns of more than 30 percent of the body surface area, involvement of this increased vascular permeability exists throughout the vascular tree, although

most pronounced in the area of the burn (Moncrief 1973:444). Moore (1970:1249) describes a burned patient accumulating a parasitic (i.e., obligate) edema under the burned surface as a direct result of the thermal injury to capillary walls and local changes in the microcirculation associated with erythrocyte aggregation, hemolysis, and plasma loss into the interstitium due to pathologic sieving of large molecules. If no therapy at all is given, the burned area becomes edematous, the patient's plasma volume is reduced, and the peripheral concentration of red blood cells rises. Peripheral circulatory failure then develops, and the patient passes into a low flow state.

Replacement of fluid sequestered as a result of thermal injury is the most important goal of the initial therapy of burns involving 20 percent or more of the body surface area (Baxter 1973: 707). Since the sequestration of isotonic fluid into the burn wound has been shown experimentally to occur within 12 to 18 hours after injury, Baxter et al. (1973:707) advocate the use of Ringer's lactate at a rate of 4 cc per kilogram of body weight per percent of body area burned to be given over a 24 hour period. One-half of the calculated volume of Ringer's lacate is given in the first eight hours after burning, one-fourth is given in the second 8 hour period, and the remaining one-fourth in the third 8 hour period. The calculated volume is based on total percent body burn, not on a maximum of 50 percent of the body surface as many formulae have previously recommended.

In applying a resuscitation method to children, Bruck, Asch, and Pruitt (1970:658) describe two-problems. The first is the common error of underestimating the fluid requirements of the burned child. The error occurs because the child's total body surface area is much larger in proportion to weight than that of the adult. The second problem is that of estimating the depth of the wound in scald burns in young children. The authors feel that all too frequently, areas which first appear to be second degree burn are found to be full thickness by the third to fifth postburn day. Frequently, patients with very deep burns will require additional resuscitation fluids. Baxter et al. (1973:707) state that the younger child, below the age of 2 years, is often said to require more fluids than anticipated by formula prediction. In his review of the total volume of fluid required by 150 patients age 0 to 2 years, this indeed is true. The increase is accounted for in the fact that the water losses in this age group are often in excess of 100 cc per pound per day, and therefore a maintenance volume of water approaching the amount of crystalloid solution necessary for the initial losses into the burn wound is required. These two points, in large part, account for the difference in total quantity of fluid necessitated by the younger child.

Bruck, Asch, and Pruitt (1970:658) report that one can anticipate an increase of 10 to 15 percent, and in some children, 20

percent, of body weight during fluid resuscitation which is followed by a daily weight loss during the next 10 days. Moore (1970:1249) predicts a 10 percent weight gain.

Although no information on post-resuscitation hypertension in pediatric burn patients is available, Ledgerwood and Lucas (1974: 531) report the problem is a frequent occurence following successful resuscitation of severely injured adult patients with hemorrhagic shock. The combined systolic and diastolic hypertension, 150/100 mm Hg, was sustained for a minimum of 6 hours in 20 of 33 reported cases. The cause of the post-resuscitative hypertension appeared to be related to an acute hypervolemic state associated with mobilization of previously sequestered sodium and water and a delay in recovery of adequate renal tubular function.

An example of an adult who has been overinfused with fluid and whose blood volume is 7 liters instead of the normal 5 liters has been presented by Guyton (1971:279). This increases the mean systemic pressure to more than double normal; it more than doubles the cardiac output, increases the arterial pressure very greatly, and therefore, increases the rate of loss of extracellular fluid from the body.

Hypertension Associated with Cardio-vascular Failure

Joshi (1970:2130) presents an analysis of clinicopathological data of 7 fatal cases of burned children which shows that congestive heart failure was a major factor in their deaths. The patients

demonstrated infected burns, prolonged clinical course, anemia, epidoses of hypokalemia and hyperkalemia, systemic hypertension, and hypoproteinemia. The study indicates that cardiomegaly and congestive heart failure occur with some frequency in children with burns. The actual pathology was not conclusively determined, but several factors, such as focal inflammatory and degenerative lesions of the heart, increased cardiac output, systemic hypertension, prolonged clinical course, electrolyte imbalances, hypoproteinemia, and malnutrition may have played a role. Although the exact mechanism is unknown, the presence of the clinical observation of a decreased cardiac output, unexplained by any change in blood volume, suggests a direct myocardial effect of thermal injury (Moncrief 1973:444).

Brand, Cowgill and Lefer (1969:216) demonstrated a filterable myocardial depressant factor circulating in the blood of burned animals which produces a decreased cardiac output in unburned animals through a crossperfusion technique. Although the ultimate response in shock is one of decreased cardiac output and hypotension, a compensatory mechanism may be a transient rise in blood pressure (Schwartz 1974:133). Inability of the cardiovascular system to maintain the continued high cardiac output necessary to meet the metabolic requirements of the thermally injured results in peripheral vascular constriction in the tissues still able to respond (Clowes et al. 1970:663). This constriction could produce a transient hypertension.

Hypertension Associated with Electrolyte Imbalances

After injury with all manner of burning agents, a sequence of fluid shift occurs (Batchelor, Sutherland, and Clover 1965:130). As the capillaries become more permeable, increasing volumes of protein and electrolyte fluid leave the intravascular compartment and enter the interstitial compartment of the extracellular space to form the burn wound edema. Baxter (1971:7) has demonstrated that edema fluid, collected by suction from tubes inserted into the burn wound, reveals a sodium concentration in the burn edema fluid of between 125 and 144 mEq/1. The sodium:potassium ratio is similar to that of the composition of extracellular fluid with similar findings at any interval between 6 and 30 hours postburn. Since the fluid being lost is basically isotonic, the replacement should also be isotonic. Ringer's lactate furnishes the following:

Sodium	130 mEq per liter
Potassium	4 mEq/per liter
Chloride	109 mEq/per liter
Lactate	28 mEq per liter

Plus 80-100 ml free water per liter (Monafo 1971:1).

Baxter (1971:7) states that Ringer's lactate is the fluid of choice because it most closely approximates the chemical composition of the fluid being lost.

Sodium

Warden et al. (1973:420) describe the adequately hydrated burn patient as one who maintains a serum sodium level of 132 to 138 mEq per liter. A lower serum sodium level indicates over administration of sodium-free solutions while hypernatremia indicates a requirement for additional electrolyte-free water.

Sodium ions represent approximately 90 percent of all the extracellular cations. Therefore, sodium is the single most important ion that needs to be regulated. A specific mechanism for regulating its concentration is vested in the kidneys and the adrenal cortex. The precise amount of sodium reabsorbed is regulated by the concentration of aldosterone, a hormone secreted by the adrenal cortices, in the body fluid (Guyton 1971:416). One of the most important of the stimuli that increase the rate of aldosterone secretion is low sodium concentration in the extracellular fluid. A 5 percent decrease in sodium concentration continued over a period of several days approximately doubles the rate of aldosterone secretion. Conversely, a similar increase in sodium concentration reduces aldosterone secretions to levels far below normal (Guyton 1971:416).

After 18 to 24 hours postburn, when most or all of the calculated amount of Ringer's lactate has been infused and signs of adequate resuscitation are less than optimal, small amounts of plasma are given slowly to restore the circulating volume to an acceptable level (Baxter 1971:7). If the fluid needs are not met, several problems, including hypernatremia, will occur. Significant abnormalities of serum sodium concentration generally result from inadequate water administration, heart failure during fluid mobilization, exudative losses from the open wound replaced with water alone, or when sepsis is responsible for circulatory failure. Hypernatremia (above 150 mEq per liter) may result from inadequate water administration.

Most frequently, serum hyponatremia of clinical importance occurs between the third and tenth days postburn when fluid is being mobilized (Baxter 1967:747). This is because of treatment modalities such as hydrotherapy and local wound care as well as by the person drinking large quantities of free water.

The regulation of fluid and electrolyte balance consists of (a) preservation of optimum total body concentration and (b) distribution of chemically active particles in the intracellular and extracellular fluids. The most important organ regulating balance is the kidney. A number of mechanisms act on the kidney to cause excretion of greater or less amounts of water and to cause reabsorption or release of varying amounts of sodium, potassium, chloride, bicarbonate, and hydrogen ions (Reed and Sheppard 1971:10). Similarly, the secretion of renin increases the secretion of aldosterone.

Renin is a hormone secreted by the juxtaglomerular cells of the kidney. With severe burns and/or trauma, excessively large quantities of this hormone are secreted in the blood and circulate throughout the body. The renin reacts to cause formation of angiotensin which causes vasoconstriction and increased secretion of aldosterone. Though a tremendous amount has been written about this renin mechanism, its significance has been proved only under special conditions rather than in the vast majority of cases (Guyton 1971:308).

When sodium concentration falls to a low value, renin is formed by the kidneys, which in turn increases the concentration

of angiotensin in the blood. The angiotensin increases the secretion of aldosterone, which causes increased reabsorption of sodium in the extracellular fluid until its concentration can rise back to normal. Conversely, a high level of sodium diminishes renin secretion and the opposite sequence of events leads to decreased extracellular sodium. This mechanism could also explain the increased sodium retention by the kidneys when the blood volume or cardiac output falls below normal, for diminished blood flow through the kidneys increases the formation of renin, which causes (a) sodium retention, (b) increased extracellular and blood volumes, and (c) increase of the cardiac output back toward normal (Guyton 1971:417).

Warden et al. (1973:420) present the data from 51 patients, representing a total of 10.9 percent of the admissions to a burn unit in an 18 month period, who developed hypernatremia with a serum sodium level above 145 mEq per liter. Of these 51 patients, 60.8% percent had inadequate replacement of insensible fluid losses, and 31.3 percent of the patients developed hypernatremia during septic states. A small group of patients developed hypernatremia secondary to a solute diuresis with uncontrolled diabetes mellitus or supranormal caloric feedings. Two patients were identified with an apparent derangement in the antidiuretic hormone mechanism.

Hypernatremia may produce hypertension as the body shifts fluid into the circulating blood volume in an effort to dilute the sodium concentration. Hyponatremia is a less likely source of hypertension, but it may also occur as the body ceases to excrete sodium

and water, even though the ultimate goal would be to decrease sodium excretion. However, this hypertension would be very transient as the excess free water in the circulating blood volume moves out of the vasculature and into the intracellular spaces in an effort to raise the serum sodium levels. Prolonged fluid retention which results in increased blood volume stresses the heart and will eventually lead to congestive heart failure.

Reed and Sheppard (1971:276) state that sodium excretion may rise to twice normal during the diuretic phase of recovery from burns and begins to approach normal at about a week after the injury. Antoon, Volpe, and Crawford (1972:609) list hyponatremia as the etiology of encephalopathy in 4 of the cases from a total of 20 pediatric burn patients who developed encephalopathy. Systemic bacterial sepsis is often accompanied by a precipitous drop in serum sodium concentration. This sudden change is poorly understood, but usually accompanies loss of extracellular fluid as either interstitial or intracellular sequestration (Shires, Carrico, and Canizaro 1973:145).

Potassium

Aldosterone secretion is also increased in response to high potassium levels. Tubular epithelial cells both reabsorb and secrete potassium ions. The potassium secretion phase, the phase affected by aldosterone, takes place as an exchange mechanism across the tubule. As sodium ions are reabsorbed from the lumen into interstitial

fluid, potassium ions are secreted in the other direction, to maintain electrical neutrality (Reed and Sheppard 1971:127). Elevation of the serum potassium concentration by approximately 10 percent, a change of only 0.4 mEq/liter, doubles the rate of aldosterone secretion. In addition to the aldosterone mechanism for control of serum potassium ions, an increase in this concentration also has a direct effect on the distal tubules and collecting ducts to cause potassium secretion into the tubular fluid. This has been postulated to result from the following mechanism: increased potassium concentration in the extracellular fluids causes increased potassium ion concentration in the epithelial cells of the distal tubules and collecting ducts. This in turn provides increased quantities of potassium to diffuse into the tubules. Under the influence of the electronegativity in the tubules, the potassium is passively secreted (Guyton 1971:416).

Baxter (1967:747) cites electrolyte abnormalities as commonly seen throughout the burn course and emphasizes that the importance of these changes should not be minimized since their occurrence represents a serious systemic problem. Hemolysis and tissue destruction may produce a mild hyperkalemia during the first several days, but this does not become clinically significant if a urine volume of 20 to 40 cc per hour is maintained.

Hypokalemia is always a possibility as extracellular potassium moves back into the cells. This spontaneous re-entry, combined with a measure of urinary depletion due to diuresis, reduces serum potassium levels to a point that oral or intravenous potassium is needed (Reed and Sheppard 1971:293). Shuck and Moncrief (1969:3) state that mobilization of edema fluid from the interstitial spaces begins approximately 3 or 4 days after injury but may not be complete until 7 to 8 days postburn. According to Moore (1970:1249), the onset of diuresis indicates that the injured capillaries have healed, and that plasma protein concentration has been maintained so that normal sieving can return with reabsorption of the edema fluid and its excretion by the kidney. Due to the leakage from the burn wound and to the diuresis, potassium is excreted in large amounts. Baxter, Marvin, and Curreri (1973:707) state that, beginning 36 to 48 hours postburn, dosages of 80 to 160 mEq of potassium per day are often required to maintain a normal serum potassium concentration.

Joshi (1970:2130) in an analysis of clinicopathological data of 7 fatal cases of burned children showed that, although congestive heart failure was a major factor in the deaths, the patients also had infected burns, prolonged clinical course, anemia, episodes of hypokalemia and hyperkalemia, systemic hypertension, and hypoproteinemia. All the patients exhibited a few episodes of hypokalemia and hyperkalemia, with 2 patients showing them more frequently than others. Since any deviations from normal were promptly treated, the episodes lasted only 1 or 2 days.

Calcium

Parathyroid hormone normally maintains serum calcium levels. Ionized calcium in body fluids regulates permeability of cell membranes (Reed and Sheppard 1971:132). According to Guyton (1971:419) the calcium ion is controlled by the parathyroid in the following manner: a low level of calcium in the extracellular fluids has a direct effect on the parathyroid glands to promote increased secretion of parathyroid hormone, which in turn acts directly on the bones to increase the rate of reabsorption of bone salts. This releases large amounts of calcium into the extracellular fluids and thereby elevates the calcium level to normal. Calcium is regulated by the kidneys as well, for large amounts of calcium are known to be lost into the urine when its extracellular concentration is high, while very little is lost when the concentration is low. The total mechanism for this regulation is unknown.

Although the kidneys actively secrete or retain the calcium ion, the parathyroid glands control the calcium ion concentration in the extracellular fluids, for reduced calcium concentration increases the rate of parathyroid secretion. This in turn increases bone absorption, thereby raising the calcium ion concentration to normal. Conversely, increased calcium-ion concentration inhibits the parathyroid glands, which automatically decreases the calcium ion concentration to normal (Guyton 1971:938).

Since hypocalcemia is accompanied by increased permeability of nerve cell membranes to sodium, sodium leaks across membranes with greater ease causing these membranes to depolarize easily. Continuing repetitive depolarization causes tetanic contraction of innervated muscles and can lead to death by spastic respiratory paralysis (Reed and Sheppard 1971:142).

Hypercalcemia produces rather vague symptoms at slightly elevated levels. However, with higher serum calcium levels, lassitude gives way to somnambulism, stupor, and finally coma. Schwartz et al. (1974:65) list the critical level for serum calcium as between 16 and 20 mg/100 ml, and unless treatment is instituted promptly, the symptoms may rapidly progress to death.

Earll, Kurtzman, and Moser (1966:378) present a case study of 3 consecutive patients with hypercalcemia and reversible hypertension. All three of these adults had a thyroid disease component to their illness. Moore and Smith (1963:447) experimentally produced a systolic blood pressure rise of at least 30 mm. Hg. in 9 of 19 patients by administering 15 mg. of calcium per kilogram of body weight intravenously into healthy subjects. Berliner, Shenker, and Weinstock (1972:92) present a case study of an 11 year old boy who developed hypercalcemia and hypertension after being immobilized because of third degree burns. The patient's admission calcium was 14.5 mg/100 ml. Subsequent serum calcium levels ranged between 11.7 mg/100 ml.

and 14.9 mg/100 ml. Throughout his hospitalization his blood pressure was noted to be persistently elevated with systolic pressures varying from 140 to 170 mm. Hg. and diastolic pressures varying from 80 to 120 mm. Hg. He was treated with Reserpine, hydralazine, and phenobarbital. On the sixtieth postburn day, he had a grand mal seizure with a blood pressure of 210/140 mm. Hg. and a serum calcium of 15.8 mg/100 ml. Other electrolytes were within normal limits. An intravenous pyelogram was normal; his blood urea nitrogen was 18 mg/100 ml with a serum creatinine of 0.9 mg/100 ml. X-rays of his skeleton showed generalized demineralization. A parathyroid hormone determination was within normal limits. He was treated with antihypertensive agents. The patient's blood pressure remained elevated throughout the remainder of his hospital stay with a consistently elevated serum calcium, though this was subject to considerable fluctuations. He was discharged on postburn day 110, still on oral antihypertensive agents. His serum calcium and blood pressure levels fell slowly to normal by one year after his injury.

Although the exact relationship of hypercalcemia to hypertension is unknown, Earll, Kurtzman, and Moser (1966:378) postulate two possible factors. The first is the heart's response to local hypercalcemia by which the contractile force of the heart is increased. An elevated cardiac output, in the absence of vessle dilation, would produce hypertension. The second factor is the response of the

kidneys to hypercalcemia, which is known to produce spasms in renal vessels as well as actual renal vascular damage. There is substantial experimental and clinical evidence that the calcium ion has a direct effect on vascular toxicity and that significant renal disease is not a pre-requisite to the association of hypercalcemia and hypertension.

Hypertension Associated with Renal Disease

Oliguric renal failure is the most severe renal lesion in the spectrum of renal damage. Profound oliguria with urine volumes of 100 to 200 cc. per day do not usually occur abruptly except in instances of transfusion reaction or myoglobinuria nephrosis. Most often urine volumes are between 400 to 700 cc. per 24 hours on the first day, then progressively decrease over the next 2 to 4 day period, becoming fixed at 50 to 200 cc. per day (Baxter and Shires:In Press).

Acute renal insufficiency usually follows a catastrophe such as severe trauma, shock, and transfusion reactions (Thal et al. 1971:157). Acute tubular necrosis following thermal injury has been regarded as a natural consequence but should not be, since there is no major impairment of renal function as a primary result of thermal injury. Along with the marked dynamic changes occurring in the immediate postburn period, an increase in permeability of the glomerulus in the burned patient is reflected by the appearance of large amounts of protein in the urine. A gradual and progressive increase in capillary permeability permits the loss of increasing larger molecular components from the glomerulus. By the third postburn

day, substances of a molecular weight of 150,000 are found in the urine, but after this period the lesion gradually heals. Although glomerular filtration rate may fall in the first few postburn hours, it rapidly returns to normal levels with adequate resuscitation. Tubular integrity, as reflected in the ability to reabsorb sodium and chloride, excrete potassium, and differentially handle water, creatinine, and urea, remains unimpaired. Thus, with adequate resuscitation therapy, renal function, although temporarily deranged in some aspects, can remain adequate and rapidly return to normal (Moncrief 1971:444).

Acute renal failure is usually accompanied by an oliguria of less than 20 ml. per hour or complete anuria. One of the most common causes of oliguria and renal failure is decreased renal blood flow which, in the patient with burns, may be due to 1) hypotension, 2) decreased cardiac output, 3) hypovolemia, 4) vasoconstriction of major blood vessels, 5) myoglobinuria, or 6) transfusion incompatibility (Baxter and Shires:In Press). Acute hypovolemia in the under resuscitated burn patient, severe dehydration with serum sodium greater than 151 mEq per liter, circulatory failure associated with overwhelming burn wound sepsis, and cardiac failure for whatever reason, can produce renal failure and oliguria. Feller and Archambeault (1973:75) list the most common factors contributing to oliguria or renal failure as hypovolemia, hemolysis, muscle injury which produces myoglobinuria, and sepsis.

Laboratory findings in acute renal failure generally show a serum elevation of urea nitrogen, creatinine, phosphorus, and potassium levels and a reduction in carbon dioxide, sodium chloride, and calcium plus a blood urine ratio of less than 5:1. A ratio of 20:1 or greater suggests extrarenal azotemia, while a ratio of 10:1 is borderline failure. A ratio as low as 5:1 or less, even in the presence of normal urinary volume, is indicative of incipient failure, usually on the basis of acute tubular necrosis (Baxter and Shires: In Press). Because nitrogenous wastes are completely retained by the anuric or oliguric patient in renal failure, he will be azotemic. Azotemia, the retention of nitrogenous wastes in the blood, is the syndrome of the failing kidney (Feller and Archambeault 1973:56). A characteristic of azotemia is an elevated blood urea nitrogen and creatinine, and the accumulation of phosphates and potassium in the extracellular fluid. The azotemic patient may be edematous or dehydrated, depending on whether salt and water are retained or lost. Thus, in addition to the electrolyte imbalances produced, the patient may also be volume depleted or volume overloaded. The volume overload itself may produce hypertension.

Progressive uremia occurring without a period of oliguria and accompanied by a daily urine volume of greater than 1000 to 1500 ml. per day is described by Baxter and Shires (in press) as the most frequently diagnosed clinical variant of renal failure

occurring in association with surgery and trauma. The reported incidence of high output renal failure as between 5 and 10 times more frequent than oliguric renal failure. The clinical picture shows a mounting azotemia for a mean of 10 days with a progressive decrease toward normal for the next 10 to 12 day period. The azotemia is accompanied by urine volumes which generally increase daily, reaching their height at the peak of the azotemia, and then gradually returning to a normal range (Baxter and Shires:In Press).

Cameron and Miller-Jones (1967:132) present details from a study of renal function of 110 children in Britain with burns greater than 15 percent of the total body surface area. The patients studied were divided into two groups according to the peak blood-urea concentration attained in the period following the burn injury. Those whose blood-ureas exceeded, at any time, the level of 100 mg. per 100 ml. were called "uremic." Those patients with blood urea levels below 100 mg. per 100 ml. were diagnosed as "non-uremic." This separation was made because 21 of 22 patients (15 percent) who fell into the "uremic" group died. Of the remaining 88, twelve died (14 percent). Resuscitation consisted of replacing 15 to 30 percent of the patient's body weight (half as blood or colloid) in the first 48 hours, given equally over three periods of 8, 16, and 24 hours each. Values of urine and urea output were established as normal on 34 non-uremic patients. Serial renal function studies were performed in 11 uremic and 13 non-uremic patients. Measurement

of glomerular filtration rate showed a uniform depression, maximal at 2 to 3 days postburn, and present in all cases. The authors suggest that the anuria found immediately after injury could result (1) from a nervous reflex inducing spasm of the afferent arterioles; or (2) from circulation of very toxic substances from the burned area to the kidney in sufficient quantities to cause afferent glomerular spasm, or such severe changes in capillary loops that filtration ceases immediately. The more common pattern of renal function was a decline over 48 hours to very low levels in the second and third days.

Grossman et al. (1974:807) report rhabdomyolysis with myoglobinuria from non-traumatic causes as the sole identifiable explanation for acute renal failure in 15 patients during a three year period. Although the myoglobinuria present in burn patients is due to traumatic rhabdomyolysis, the clinical picture of renal failure is comparable to that of non-traumatic myoglobinuria. Of the 15 patients in the non-traumatic group, 7 of the patients developed hyperkalemia within three days of the insult.

Severe biochemical abnormalities in myoglobinuric acute renal failure are exaggerations of abnormalities usually found in acute renal failure of other causes (in the latter group serum creatinine levels rise to an average rate less than 2 mg. per 100 ml. per day). In 12 patients in this study, it was found that during the first three days after the insult, serum creatinine levels rose

at the unusually rapid rate of greater than 2.5 mg. per 100 ml. per day. In four cases, the rapid rise in serum creatinine levels was not paralleled by a similar rise in blood urea nitrogen levels.

Creatinine is an end product of muscle creatine metabolism. Normally, about 2 percent per day of muscle stores of creatine or creatinine phosphate spontaneously and non-enzymatically dehydrate, forming creatinine. This reaction is irreversible at body temperature and pH. The concentration of creatinine per unit wet weight of striated muscle is at least 5 times that of plasma. The creatinine so formed diffuses down concentration gradients to extracellular fluid and is quantitatively excreted by the kidneys. The rapid increments in serum creatinine levels observed in rhabdomyolysis with acute renal failure could be due to the release of pre-formed intracellular creatinine into the extracellular fluid through damaged muscle cell membranes. In addition, rupture of many muscle membranes would remove the diffusion barrier that retards the appearance of newly formed creatinine in the extracellular fluid. This would cause a transient increase in the rate of rise of serum creatinine levels without an increase in total creatinine production.

The mechanism by which heme pigments cause acute renal failure is not clearly understood. Hypotheses that have been proposed include plugging of tubules by myoglobin containing casts, passive back diffusion of glomerular filtrate through damaged tubular epithelial cells, or a decrease in glomerular filtration rate. All experimental models have required the presence of dehydration for myoglobin to cause renal damage (Grossman et al. 1974:807).

Moncrief (1973:444) states that renal failure in the early postburn period has not been considered a major problem in this country. In Britain, however, it is a source of great interest and is documented to occur in a wide ranging frequency of 1.3 percent of total admissions to 15.3 percent in patients with burns involving over 15 percent body surface area. In general, resuscitation differences explain the higher rate in Britain since resuscitation is primarily with colloids in lesser quantities which are given at a slower rate. This would produce a lowered cardiac output and a decreased renal perfusion (Moncrief 1973:444). One of the complications of renal failure is hypertension associated with electrolyte imbalances and/or fluid overload.

Hypertension Associated with Hematologic Changes

Hematologic changes which occur as a result of the burn injury may include altered white blood cell activity and host defense mechanisms; blood cell alterations as a direct result of exposure to heat; altered function and life-span of red blood cells; and oxygen transport dysfunction due to hemoglobin alterations.

The leukocytes are the mobile units of the body's protective system. They are formed partially in the lymph nodes, lymphocytes and monocytes, and partially in the bone marrow, granulocytes;, but after formation they are specifically transported in the blood to different areas of the body as needed. Five different types of white blood cells are normally found in the blood. These are

polymorphonuclear neutrophils, polymorphonuclear eosinophils, polymorphonuclear basophils, monocytes, and lymphocytes (Guyton 1971:110).

Functional capability of a leukocyte depends first upon its ability to phagocytize bacteria and next upon its ability to kill and digest the phagocytized bacteria. Phagocytosis of bacteria is dependent upon a number of variables, the most important of which is coating of the bacterial cell by opsonic substances in the serum; predominantly, specific antibody and complement. In the burn injury, phagocytosis by the fixed reticuloendothelium system has been reported by Rittenbury and Hanback (1967:523) to be depressed before death occurs, but it was often elevated before that time and in survivors. Phagocytosis by subeschar leukocytes and peripheral leukocytes was reported to be normal. If phagocytosis is not blocked significantly before systemic infection occurs, intracellular destruction of ingested bacteria must be at fault (Alexander 1967:482).

Patients with burns initially have an elevated white blood cell count to 20,000 to 40,000 per cu. mm. as a result of hemoconcentration. As hemoconcentration is corrected, these findings return to nearly normal levels (Baxter 1967:915).

Many of the severe metabolic and physiological derangements accompanying large thermal injuries have been controlled by appropriate therapy after definition and characterization of these disorders by clinical observation and laboratory investigation. Disturbances

of cellular defense mechanisms, however, have been incompletely characterized, and the relationship of such changes to the subsequent development of infection has not been established (Alexander 1967:482). In an attempt to correlate the changes in serum and leukocyte lysosomal enzymes in patients with large burns with the development of infection and "burn toxemia," Alexander (1967:482) studied 13 patients with thermal injuries ranging from 30 percent to 79.5 percent. In studying the leukocyte concentration of the enzymes B-glucuronidase, acid phosphatase, and lysozyme, the concentrations of all three enzymes was decreased after burn injury. The lowest values were found between the sixth to the tenth postburn day, at a time when clinical observation has shown patients with thermal injuries to be the most susceptible to severe septic complications. The findings suggested that the decreased concentration of leukocytic lysosomal enzymes following severe burn injury is a major contributing factor to the susceptibility to major infections in these patients.

McCabe et al. (1973:155) studied the resistance status of ten burn patients with the use of sequential timed coverslip preparations on skin windows. Severely burned patients displayed uniform depletion of immune-competent cell elements, and variable depression of non-immune cell migrations.

Alexander and Wixson (1970:431) presented evidence that neutrophil dysfunction is a major determinant in the development of life-threatening sepsis after thermal injury. The occurrence of invasive bacterial infection was studied in 46 patients who

had second and third degree burn injuries involving 20 percent or more of their body surface area. The ability of blood neutrophils to ingest and kill bacteria was studied sequentially in those patients with burn injuries over more than 40 percent of their body and at random intervals in the remaining patients. The ability of neutrophils to kill ingested bacteria was found to be deficient in patients with a major burn injury. The concurrent occurrence of this dysfunction with the development of invasive sepsis was found to be highly significant.

The breakdown of the host defense mechanism occurs for reasons which are not clear. As the patient is overwhelmed by sepsis, the body, in a primary attempt to maintain blood flow will compensate by vasoconstriction which will, in turn, produce a transient hypertension (Schwartz et al. 1974:144).

Heat not only damages the vascular endothelium producing local thrombocytopenia, but also sequesters red blood cells and inflicts damage on an additional significant quantity leading to decreased survival of these cells. However, massive hemolysis occurs very infrequently in burns. A misinterpretation of the quantity of red cell destruction often occurs as a result of hemoglobinuria. Hemoglobin appears readily in the urine after hemolysis of as little as 100 cc. of red cells but clears rapidly in the first 100 to 300 cc. of urine (with large amounts of crystalloid administration). Serial measurements of red cell mass in third degree burns of 40 to 70 percent surface area have been observed

to have a maximum of 15 percent loss of originally measured red cell mass in the first 24 hours. An occasional patient with burns of extreme depth (fourth degree) may have early hemolysis of 25 percent or more of the red cell mass. On the other hand, delayed destruction may often exceed 25 to 40 percent of the red cell mass, requiring multiple transfusions beginning on the third or fifth postburn day (Baxter 1967:747). However, despite the great loss of circulating red blood cells, the hematocrit is usually markedly elevated because there is proportionately a greater loss of circulating plasma volume than red cell mass. Until the fluid sequestered as peripheral edema is mobilized into the vascular space (with resultant hemodilution), red blood cell replacement is not necessary. Mobilization of edema fluid with dilution of the blood begins approximately 3 or 4 days after injury but may not be complete until 7 to 8 days postburn (Shuck and Moncrief 1969:3).

During the interval from 48 hours to 10 days postburn, the clinical signs and laboratory values may be deceptive. Baxter (1973:42) reports there is usually a high cardiac output, slight tachycardia, declining hemoglobin concentration, and a noticeable diuresis - all attributed to the expansion of blood volume produced by mobilization of the edema fluid. The total blood volume may not be elevated but a progressive profound anemia becomes apparent.

Loebl et al. (1974:96) report that a three-part study of erythrocyte survival was carried out in burned patients to clarify

the mechanisms responsible for the rapid disappearance of red cell mass during the first week following major thermal injury. Tagged erythrocytes from burned patients and non-burned volunteers were utilized in a crossover study of red cell survival. The half-life of normal erythrocytes from non-burned donors was markedly decreased when administered to burned patients ($t\ 1/2 = 6.3 \pm 1.1$ days). The autologous half-life of the burned patients own erythrocytes was similarly decreased ($t\ 1/2 = 8.5 \pm 1.5$ days). However, when the burned patients' cells were studied in non-burned volunteers, red cell survival was found to be normal ($t\ 1/2 = 23.3 \pm 1.8$ days).

External counting of the distribution of radioisotope following injection of Cr^{51} labeled erythrocytes failed to demonstrate progressive accumulation in the burn wound or the spleen, implying that these areas are not the primary sites of red cell removal from the circulation. A randomized program of treatment with aspirin or low-dose heparin in a small series of burned patients was conducted to determine effects of such therapy on autologous erythrocyte survival. The half-life of erythrocytes in heparin treated patients (7.7 ± 1.4 days) and aspirin treated patients (6.7 ± 1.0 days) were not significantly different from the survival of autologous erythrocytes in burn patients not receiving these drugs (8.5 ± 1.5 days). This study characterized, but did not precisely identify, a humoral mechanism which results in increased erythrocyte destruction following thermal injury.

The presence of hypoxemia contributes further to the circulatory demand. Inability of the cardiovascular system to maintain the continued high cardiac output results in peripheral vasoconstriction (Clowes et al. 1970:663). Schwartz et al. (1974:494) report the varying distribution of the cardiac output to the different organs and body tissues is the body's most important defense in oxygen transport deficiency. During acute hypoxia, blood is preferentially sent to the heart and brain with flow curtailed to tissues with such low priority as the skin, skeletal muscles, and corneas as soon as hypoxia threatens. They also report that another problem of hemoglobin affinity is carbon monoxide poisoning. Patients who have been burned in closed spaces may have serious defects of oxygen transport caused by carbon monoxide displacing oxygen from hemoglobin.

The initial objective of correction of the red blood cell deficit is to maintain the hematocrit at a level slightly above normal (42 to 46 percent). This avoids excessive hemodilution by over administration of colloid, and guards against a plasma volume deficit indicated by a rising hematocrit (Moore 1970:1249). Replacement of red blood cells by transfusion is indicated when the hematocrit begins to drop at 5 to 10 days postburn in amounts necessary to maintain the hematocrit between 35 and 40 percent (Baxter, Marvin, and Curreri 1973:707). A severely lowered hematocrit results in hypoxia. Since the body's response to hypoxia is an increased cardiac output; hypertension could result.

Hypertension Associated with Elevated Renin Levels

The kidneys are capable of secreting an enzyme called renin, and the renin in turn acts on the plasma proteins to cause release of a vasoconstrictor substance called angiotensin. The angiotensin, in turn, increases total peripheral resistance and also increases aldosterone secretion by the adrenal cortex. At present there is no proof that the renin-angiotensin system plays a significant role in normal regulation of arterial pressure, but there is reason to believe that this system does cause hypertension in certain abnormal states (Guyton 1971:304).

Most traumatic events are accompanied by an increased secretion of renin, presumed to be brought about by a fall in perfusion pressure of the afferent arteroles in the renal cortex. The juxtaglomerular apparatus, whose cells are in close proximity to the afferent arteriole, is presumed to be responsive to changes in the afferent arteriole perfusion pressure, so that its cells secrete renin into the renal venous blood and lymph. The renin, in turn, activates angiotensinogen (made in the liver) into angiotensin I, which is then converted in the bloodstream into angiotensin II. Angiotensin II stimulates the adrenal cortex to secrete aldosterone. Angiotensin II (hypertensin) has a hypertensive effect of its own and is sometimes used to treat hypotension (Schwarta et al. 1974:12).

Bozovic et al. (1967:574) reported on 7 burned patients, all of whom had a plasma-renin level above the normal values (64 ± 30

ng. angiotensin per 100 ml. plasma) with a range of 203 ng. angiotensin to 6100 ng. angiotensin per 100 ml. plasma. In this group, plasma-renin activity did not seem to be negatively correlated to plasma-sodium concentration as in hypertensive subjects studied by Brown et al. (1966:153). In one patient with a plasma-sodium concentration of 157 mEq per liter, plasma-renin activity was much increased. The patient with the highest plasma-renin activity had a lowered plasma volume (hematocrit 52%). The results indicate that the sympathetic nervous system causes renin release in burned patients. However, the relationship between hypertension and renin levels is not clear.

Hypertension Associated with an Atypical Form of Disseminated Intravascular Coagulation

Many pathophysiologic changes occur in the acutely burned patient. Some are important to ultimate survival while others are seemingly unimportant to the survival of the patient. One pathological entity which may affect survival during the initial 10 day period, despite adequate fluid resuscitation, is the presence of an atypical form of disseminated intravascular coagulation as described by Baxter (1973:42). The syndrome is manifested by a progressive angiopathic hemolytic anemia, increased platelet adhesiveness with a tendency toward thrombocytopenia, and a secondarily enhanced production of consumable clotting factors. In patients with burns involving more than 40 percent total body surface area, a detectable

level of fibrin split products in the circulating blood is present. While the role of fibrinogen degradation products has not been settled, they may be of great importance in the formation of burn edema, in the hematologic and rheologic aspects of thermal injury, and in the progression of thrombosis in the injured skin.

Feller and Archambeault (1973:296) report fibrinogen levels are universally elevated in the burned patient, sometimes reaching levels as high as 700 mgm percent by 5 days post-injury. Baxter (1973:707) reports fibrinogen levels rapidly increase to twice normal levels, with a gradual return to normal by the tenth postburn day. These changes in the clotting profile are not diagnostic signs of any of the profound intravascular clotting abnormalities that might be suspected from reported animal studies. The observed changes can easily be accounted for by the thrombosed, damaged blood vessels in the burn wound, and the extravascular coagulation often visible in burn edema fluid.

Since the extensively burned patient suffers extensive vascular damage due to heat coagulation of skin and subcutaneous tissue and sometimes deeper tissues, one might anticipate that in the junctional areas of injury and spared tissue there would be vascular damage with release of coagulant activators and thrombosis. This fibrination, if acute and extensive, could result in the depletion of coagulation factors or consumptive coagulopathy manifested

by a reduction of fibrinogen and platelets. Secondary fibrinolysis would result in an appreciable increase in fibrin degradation products (Meyers 1972:404).

Fibrin split products were measurable by Meyers (1972:404) in 77 patients with burns and 40 normal individuals who were the controls. The data indicated no good correlation between the percentage of surface burns and the levels of fibrin split products.

Curreri, Wilterdink and Baxter (1974:157) instituted a prospective patient study to test the effectiveness of prophylactic systemic anticoagulation in preventing abnormal elevations of fibrin split product concentration in the immediate postburn period. Thirty-one patients with greater than 30 percent total body surface burns, who had no gross contraindications for systemic anticoagulation, were randomly assigned to one of 3 patient groups. One group was administered heparin, 5,000 units subcutaneously every six hours during the first two postburn weeks. A second group received aspirin, 10 grains per rectum, during the same time period. A control group of patients received neither heparin nor aspirin. Failure to alter fibrin split product concentration in the patient population receiving heparin therapy was somewhat surprising. It was possible that inadequate anticoagulation was achieved though most patients exhibited simultaneous prolongation of their partial thromboplastin time. More likely, the elevated fibrin split product elevation seen in the postburn period was believed to not be secondary to abnormal intravascular coagulation but rather secondary to coagulation in the extracellular edema fluid.

In view of this data, a further study was indicated to assess the actual location of the fibrinogen and the fibrin. Utilizing a 40 percent flame-burned canine model, Curreri et al. (1975:86) collected serial samples of burn-wound edema fluid and simultaneous plasma samples for 26 hours postburn, following the injection of tagged fibrinogen. The data obtained in the experiment was not compatible with postburn intravascular coagulation and strongly suggested that the observed increase in fibrin split product concentration in the plasma of burned patients is secondary to extravascular degradation of fibrinogen and fibrin. These results were explained by the following hypothesis. Increased capillary permeability to fibrinogen and fluid as a result of the burn injury results in the extravascular accumulation of fibrinogen in the wound. Plasmin degradation is rapidly activated, resulting in the early appearance of fibrin split products in this extravascular site. As capillary repair occurs at 24 hours after burn, the large molecular weight soluble fibrin and fibrin complexes become entrapped in the edema fluid. Spontaneous uncoupling of soluble fibrin complexes with D fragment in the edema fluid would produce free, small molecular weight D fragment which can freely pass back into the intravascular space and then again complex with circulating soluble fibrin, thus resulting in increased plasma fibrin split product concentration at 24 hours.

Platelet adhesivenss is markedly increased immediately after burn, often increasing to 95% from a normal of 45-55%. Studies of platelet half-life in 12 patients with burns involving 30 to 65 percent of the total body surface area (Shires et al. 1970:308), suggest a shortened survival time, but the extreme adhesiveness of the thrombocytes may represent an apparent decrease in survival associated with sequestration of platelets aggregates which later break up.

Thrombocytopenia is the presence of a very low quantity of platelets in the circulatory system. Persons with thrombocytopenia have a tendency to bleed from many small capillaries rather than from large vessels. As a result, small punctate hemorrhages occur throughout all the body tissues. Ordinarily, excessive bleeding does not occur until the number of platelets in the blood falls below a value of approximately 70,000 per cubic mm. rather than the normal of 150,000 to 350,000 (Guyton 1971:143).

Feller and Archambeault (1973:295) discuss platelet abnormalities seen in a study of burned patients. Assuming the severely burned patient had a normal platelet count prior to admission, about 5 days postburn the platelet count fell to 64 percent of normal for those patients who survived and to 50 percent of normal for those who died. About 30 days postburn, the count rose to 180 percent of normal for the patients who survived. If the platelet count did not return to normal in the same period of time, the

patients died. In those who died, a platelet count of less than 100,000 per cu. mm. (range 1,000 to 96,000) occurred one to two days prior to death.

Hypertension Associated with the Use of Hexachlorophine

The term "burn encephalopathy" has appeared frequently in the literature to describe the burned patient who develops stupor, coma, confusional states, muscle twitching, convulsions, cerebral deficits, chorea, athetosis, hyperkinesia, and cerebral edema (Larson 1968:63) which produces hypertension and/or seizures. This condition has been found predominantly in children.

A survey of possible causation of this syndrome in burned children by Larson (1968:63) implicated hexachlorophene treatments as a possible etiologic agent. This led to a study that consisted of (1) investigating the blood levels of hexachlorophene of patients subjected to various treatments with hexachlorophene preparation and (2) studying the effects of hexachlorophene preparation on experimental animals.

Eight patients were studied with regard to hexachlorophene levels in the serum following contact of burn wounds with 3 percent hexachlorophene detergents. Three of the patients were children who were admitted to the Shriners Burns Institute with a history of convulsive seizures prior to admission. All of these children had undergone daily washing with 3 percent hexachlorophene detergent

in other institutions prior to admission to the Institute. Serum levels of hexachlorophene were determined five to eight days following the last wash, and 4 to 8 µg. of hexachlorophene per ml. of serum were found. One child exhibited bizarre neurological symptoms; a cerebrospinal tap showed 1.7 µg. of hexachlorophene per ml. of cerebrospinal fluid. The other 5 patients studied were adults who had received 3 percent hexachlorophene washes and who had ranges of 9.3 to 74 µg. per ml. of serum. The patient with the highest level was a 19 year old male with burns over 50 percent of his body. At the time of admission to the Institute, one leg was grafted and the other was washed with 3 percent hexachlorophene detergent for 4 days. On the fourth day of washing, the patient had convulsive seizures with a serum hexachlorophene level of 74 µg. per ml. serum. The hexachlorophene was discontinued; the patient's symptoms cleared within 3 days, and he had no further difficulties.

In another study, 150 rats were subjected to 40 percent scald burns and divided into groups with different treatments. All rats treated with hexachlorophene died. On autopsy, cerebral edema was the most common finding. Thus, there does appear to be a relationship between the use of hexachlorophene and the incidence of hypertension.

Hypertension Associated with Psychological Factors

Holter and Friedman (1969:680) report a severe burn in a child is an extremely stressful experience for both the child and his family. Not only is the physical recovery slow and painful, but the psychological trauma has long-lasting effects.

Post discharge studies by Woodward (1959:1009) and Woodard and Jackson (1961:316) of the children who had recovered physically from severe burns have revealed that emotional disturbances exist in approximately 80 percent of the children and 60 percent of their mothers. The majority of these mothers thought the disturbances in their children were the result of the burn experience, pain, and the separation from home during the hospitalization. However, Long and Cope (1961:1121) report a high incidence of psychopathology in the family unit antedating the burn incident and Vagliano, Hart, and Singer (1964:753) further suggest that chronic relationship problems may become overtly manifest only at the time of the children's burns. Work by Holter and Friedman (1968:693) also emphasizes the importance of considering the "battered child syndrome" in children with burns.

Borland (1967:693), in his report on prevention of childhood burns, concludes:

> The key to successful intervention in the process leading to childhood accidents is awareness of high risk groups, and recognition of acute stress episodes within chronically stressful families... Since each accident arises out of a unique and complex set of emotional and physical circumstances, universally applicable methods of accident prevention are very difficult to introduce. The case study

> approach, however, can be most advantageous, inasmuch as recognition of unusual or unexpected behavior or of possibly adverse social or physical environmental conditions can be a means to anticipation of acute stress episodes.

Holter and Friedman (1969:680), in assessing the emotional makeup of the families of severely burned children, found that 10 of 13 families studied had major psychological and social problems within the family units prior to the burn incidents. In these 10 cases, the gross emotional disturbances within the families appeared to have propelled the children into tragic situations resulting in severe burn. The cases reported illustrated that most childhood burns may be categorized as reflecting (1) a true accident, (2) a situational crisis, or (3) child abuse. Thus, the overall management of burned children and their families is determined, in part, upon this type of etiologic considerations.

Seligman (1974:41) has proposed a system of classiciation of burned children designed to identify children's normal and pathological response to severe burns and to facilitate research into the prevention of serious psychological complications. In her proposed psychiatric classification scheme for assessing burned children, Seligman divides the data collection into 3 categories: child-related, burn-related, and environment-related. The goal of such a system is to find the norms for children undergoing major stress; those who deviate from these norms can then be identified for more thorough observation. Perhaps then the health team members will come closer to knowing who requires psychiatric care and how limited psychiatric facilities may best be utilized.

In an effort to isolate possible emotional factors in the survival of severely burned children, Seligman, Carroll, and MacMillan (1971:655) first identified, statistically, the more obvious factors affecting survival rate among 152 patients at their hospital. They then contrasted survivors and non-survivors having approximately the same known expectation of survival. Distribution of the 152 patients by percent total body burn and age was studied. One hundred and eight patients had less than 40 percent total body burn and all but 2 survived. Of the remaining 44 having total body burns 40 percent and over, 27 lived and 17 died. In the 40-49 percent total body burn category 18 of 19 survived; in the 50-59 percent category 6 of 16 survived; in the 60 percent and over 3 of 9 survived and 5 of these were over 69 percent and none survived. Thus they suggest that it appears emotional factors might be most critical for survival with children suffering burns of from 40 to 70 percent of their body. No survival trends related to age and sex were found except that no child under the age of one year was admitted who had 40 percent or over total body burn and there were no male admissions in the age category of 3 to 6 years.

For further investigation, they selected 7 children with 40 to 59 percent total body surface burn for a retrospective study of hospital course with emphasis on emotional factors and family histories. Historical data included family intactness; whether parents and siblings were dead, alive, or absent; crises; changes

or identifiable events; whether the injury was of the child's own doing; parents presence or absence at the burn moment; parent-child relationship; level of psychosexual development and ego strength.

During hospitalization data considered was behavioral responses, psychosomatic responses, predominant type of anxiety, defenses, symptoms and affect, body concern, and parental responses. Several possible trends were noted. Survivors had greater quantity and more severe quality of clinging and demanding behavior with more protest. The non-survivors had withdrawn, despair, or detached type behavior. All survivors and none of the non-survivors had psychosomatic responses. Three survivors had hypertension.

This study points toward three possible trends for survival: a poorer prognosis in preadolescents who lose a parent of the same sex and are close emotionally to the surviving parent; a better prognosis in those with protest behavior and psychosomatic responses; and a better prognosis for children with hopeful parents.

Seligman, MacMillan, and Carroll (1971:84) studied 4 cases of children who had suffered major burn injury. Significant factors that emerged included unconscious motivation and early parent loss in some parents of burned children, an unexpected finding. Positive factors for survival were coping mechanisms of denial, withdrawal (although this factor seemed contraindicated in a study done by the authors in 1974), splitting of the ego, somatic responses (hypertension, ulcers, and "seizures"), and hope in parents and/or staff.

It was found that somatic responses (hypertension, ulcers, and "seizures") occurred in survivors and not in non-survivors.

Summary

A review of the literature available on hypertension as a complicating factor in poliomyelitis was presented since the clinical findings in both poliomyelitis patients and pediatric burn patients with hypertension appears to be similar. Although the exact etiology of the hypertension which develops in children with burn injuries is not known, there are several clinical states which, by themselves, might produce hypertension. These clinical states have been reviewed and the pathophysiology involved has been presented. When available, actual case studies related to the causes were presented. The nursing implications related to the care of the pediatric burn patient, in general, were presented.

CHAPTER III

PROCEDURE FOR COLLECTION AND TREATMENT OF DATA

The purposes of this study were to determine the incidence of hypertension in the pediatric burn population, to determine the cause of hypertension in the pediatric burn patient, and to describe the pediatric burn population with hypertension. To meet the needs of the design of the study, the study was retrospective in nature (Abdellah and Levine 1965:214) and conducted by the descriptive nonexperimental design (Abdellah and Levine 1965:40). Demographic data was collected on all patients who met the criteria of the study; in addition, clinical data was collected on all hypertensive patients. An attempt was made to collect data regarding renin and angiotensin levels in the patients. However, due to an inadequate number of samples, this data was not included in the study. Due to the retrospective nature of the study, the researcher was unable to collect accurate data regarding the depth of the burn injury. For this reason, no data regarding depth of burn injury was included.

Setting

The total population consisted of all patients admitted to the pediatric burn service of Parkland Memorial Hospital, a 797 bed county teaching hospital located in Dallas, Texas, from June, 1965 through June, 1975. Prior to October, 1971, the children were admitted to a general pediatric floor and placed in private isolation rooms. After that time, the children were admitted to a 10 bed pediatric burn intensive care unit.

Population

The total number of patients admitted to the pediatric burn service over the 10 year period was 984. Of the total, 159 patients had small infected burns and were not included in this study. The charts of 78 children could not be located. A primary diagnosis of burns was made in 747 patients who were admitted to Parkland Memorial Hospital between June 1, 1965 and June 1, 1975. Of these, 9 were eliminated from the study because of preexisting disease states as follows: 6 patients had preexisting central nervous system disorders characterized by seizures and 3 patients had congenital cardiac disease. Of all the children admitted to Parkland Hospital, 738 met the following criteria: the population consisted of the children who were admitted to Parkland Memorial Hospital with a primary diagnosis of burns between June 1, 1965 and June 1, 1975; and the children had no preexisting renal, cardiovascular, or central nervous system disease.

Hypertensive children were those who met the above criteria and who maintained an elevated blood pressure for a period of 24 hours or more. Elevated blood pressure is the mean diastolic blood pressure by age as described by Mitchell et al. (1975:1) plus 20 mm. Hg. (Appendix A.) The normal blood pressure values are as follows: 0 to 6 months, 45 mm. Hg.; 6 months to 3 years, 64 mm. Hg.; 4 to 5 years, 65 mm. Hg.; 6 to 13 years, 70 mm. Hg.

Tool

In designing the forms necessary for the data collection, two methods were used. The researcher developed a form which was considered adequate by the members of the thesis committee and the statistician for data collection. Demographic data (Appendix C) was collected from the charts of patients who met the criteria of the study and included admission date, age, sex, race, percent of total body surface area burn, type of burn, family history of seizures or high blood pressure, total fluids given during the first 24 hours, adequacy of resuscitation, administration of Phisohex baths and number of days of use, a clinical diagnosis of sepsis and postburn day of diagnosis, survival or death, family situation as available, area of body burned, and the presence or absence of hypertension. Data (Appendix D-1, D-2) was collected on all hypertensive patients and included: diastolic blood pressures every 4 hours, maximum daily body temperatures, 24 hour intake and output, daily weights, and daily laboratory data consisting of white blood cell counts, hemoglobin, hematocrit, platelets, blood urea nitrogen, creatinine, sodium, potassium, chloride, calcium, and presence or absence of the sickling trait.

A preliminary study was conducted on 10 patients who met the above criteria for hypertensive patients except that the patients were admitted before June 1, 1965. After this preliminary study

was completed, the researcher, the thesis committee, and the statistician met and reviewed the tool. Several changes were made which resulted in the forms being accepted as suitable for the study (Appendix C, D-1, D-2).

Data Collection

Agency permission for use of the charts of all pediatric burn patients admitted from June, 1965 through June, 1975 was obtained, in writing, from Parkland Memorial Hospital (Appendix E). The log books which were kept as a record of all admissions to the general pediatric unit before October, 1971, were obtained and a list of the names of all patients with a primary diagnosis of burns was compiled. A list of all patients admitted to the pediatric burn intensive care unit from its opening through June, 1975 was obtained. The children's medical records were then reviewed. Demographic data was collected on all available charts of patients who met the criteria for this study. Additional data was collected on the patients who met the hypertensive criteria. The information was coded and all data was placed on the computer by personnel in the Medical Computing Resources Center at The University of Texas Health Science Center, under the supervision of Dr. Richard Browne.

Treatment of Data

All data was key-punched and computerized. It was then analyzed, using two methods, which reflect frequency and significance. The first method, used for the demographic data, was the chi-square test, a non-parametric statistical test of significance based on the chi-square distribution as described by Abdellah and Levine (1965:699). The test was used to analyze the significance of differences between the hypertensive and the non-hypertensive groups as compared in terms of qualitative variables. Frequency distributions consisted of counts of the number of study subjects in each group found to possess each of the scale values of the demographic variables measured. The test was performed by calculating theoretical frequencies for each scale value, subtracting the theoretical from the actual frequencies, squaring the difference, dividing by the theoretical frequency, and then summing up all the quotients. This sum was the computed value of chi-square for the sample data. The larger the computed value of chi-square, the smaller was the probability that the difference in the frequency distributions compared were due to random sampling. The variables present in this study were analyzed independently, in groups, and totally for a test of chi-square significance. Comparision of the distribution of the variables between the hypertensive and the non-hypertensive group was undertaken in an attempt to determine which factors or group of factors might cause hypertension and

which factors do not appear to be related to the hypertension. Chi-square contingency table analyses are an established method which can be used for the purpose of comparing distribution and frequency.

The second method used for analysis of laboratory data was the Kendall Tau-B correlation coefficient. A summary measure called r that varies between -1 and +1 was used as a measure of the degree of relationship between the variables studied. The closer the computed values or r was to +1 or -1, the higher was the degree of relationship among the variables studied (Abdellah and Levine 1965:699). Dr. Richard Browne supervised these analyses.

Summary

The total number of pediatric patients admitted to Parkland Memorial Hospital with a primary diagnosis of burns from June, 1965, through June, 1975, was 984. Of this number, 738 patients met the requirements of this study. Hypertensive criteria, which consisted of a sustained diastolic blood pressure of 20 mm. Hg. above the normal limits for the age group for a period of at least 24 hours, was met by 77 patients. Data was then collected on these patients from their medical records and placed on the computer. Analysis of the data was conducted using the chi-square test.

CHAPTER IV

ANALYSIS OF DATA

In describing the population of pediatric burn patients with hypertension, a comparison between the population with hypertension and the population without hypertension was necessary. For this reason, the demographic data (Appendix C) was collected on all patients who met the criteria of the study. The following information reflects the demographic data obtained from the total population of 738 patients who met the criteria of this study (Column I). It also reflects the demographic data of the 661 non-hypertensive patients (Column II) and the 77 hypertensive patients (Column III). The incidence of hypertension was 10.4 percent.

Over the ten year period, the distribution of patients admitted by month was uneven. In the ten year period, the largest number of patients, three hundred and fifteen or 42.7 percent, were admitted in the first four months of the year. June was the lightest month with forty patients (5.4 percent) being admitted. There was no statistical difference between the hypertensive and the non-hypertensive group with relation to month of admission (Table 3).

TABLE 3

ADMISSIONS BY MONTH OVER TEN YEAR PERIOD

Month	Column I Total Population		Column II Non-Hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
January	86	(11.7)	72	(10.9)	14	(18.2)
February	94	(12.7)	81	(12.3)	13	(16.9)
March	70	(9.5)	61	(9.2)	9	(11.7)
April	65	(8.8)	60	(9.1)	5	(6.5)
May	46	(6.2)	45	(6.8)	1	(1.3)
June	40	(5.4)	34	(5.1)	6	(7.8)
July	62	(8.4)	52	(7.9)	10	(13.0)
August	54	(7.3)	49	(7.4)	5	(6.5)
September	43	(5.8)	39	(5.9)	4	(5.2)
October	55	(7.5)	50	(7.6)	5	(6.5)
November	59	(8.0)	59	(8.9)	0	(0.0)
December	64	(8.7)	59	(8.9)	5	(6.5)
Total Number of Patients	738	(100.0)	661	(100.0)	77	(100.0)

The number of patients admitted by year (Table 4) showed an increase in the number of patients being admitted each year but with a very low patient census in the first half of 1975. The hypertensive group does not differ significantly from the non-hypertensive group in month of admission. However, the incidence of hypertension in the patients by year of admission is markedly higher in the years 1968, 1971, and 1972.

TABLE 4

ADMISSIONS BY YEAR OVER A TEN YEAR PERIOD

Year	Column I Total Population		Column II Non-Hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
June 1 - December 31, 1965	32	(4.3)	30	(4.5)	2	(2.6)
1966	61	(8.3)	55	(8.3)	6	(7.8)
1967	60	(8.1)	56	(8.5)	4	(5.2)
1968	63	(8.5)	43	(6.5)	20	(26.0)
1969	79	(10.7)	77	(11.6)	2	(2.6)
1970	71	(9.6)	67	(10.1)	4	(5.2)
1971	75	(10.2)	63	(9.5)	12	(15.6)
1972	101	(13.7)	85	(12.9)	16	(20.8)
1973	81	(11.0)	74	(11.2)	7	(9.1)
1974	97	(13.1)	94	(14.2)	3	(3.9)
January 1 - June 30, 1975	17	(2.3)	16	(2.4)	1	(1.3)
Total Number of Patients	738	(100.0)	661	(100.0)	77	(100.0)

The total population was fairly well distributed by age group (Table 5) with a slightly larger number of total patients in the 13 to 15 month old group (112 patients; 15.2 percent of admissions) and in the 19 month to 2 year old group (145 patients; 19.6 percent of admissions). Thus, 34.8 percent of the total patients were 13 months to 2 years of age. In the non-hypertensive group,

36.9 percent (244 patients) were in the 13 month to 2 year old age group. In the hypertensive group, there were no patients in the 13 to 18 month old age group but 16.9 percent (13 patients) were in the 19 month to 2 year old age group.

TABLE 5

ADMISSIONS BY AGE

Age	Column I Total Population		Column II Non-Hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
0-3 months	9	(1.2)	8	(1.2)	1	(1.3)
4-6 months	23	(3.1)	19	(2.9)	4	(5.2)
7-9 months	37	(5.0)	36	(5.4)	1	(1.3)
10-12 months	47	(6.4)	46	(7.0)	1	(1.3)
13-18 months	112	(15.2)	112	(16.9)	0	(0.0)
19 months-2 years	145	(19.6)	132	(20.0)	13	(16.9)
3 years	68	(9.2)	62	(9.4)	6	(7.8)
4 years	56	(7.6)	50	(7.6)	6	(7.8)
5 years	40	(5.4)	34	(5.1)	6	(7.8)
6 years	35	(4.7)	27	(4.1)	8	(10.4)
7 years	33	(4.5)	25	(3.8)	8	(10.4)
8 years	39	(5.3)	36	(5.4)	3	(3.9)
9 years	22	(3.0)	19	(2.9)	3	(3.9)
10 years	33	(4.5)	26	(3.9)	7	(9.1)
11 years	17	(2.3)	14	(2.1)	3	(3.9)
12 years	16	(2.2)	10	(1.5)	6	(7.8)
13 years	6	(0.8)	5	(0.8)	1	(1.3)
Total Number of Patients	738	(100.0)	661	(100.0)	77	(100.0)

There was no significant difference by sex between the hypertensive and the non-hypertensive group. No significant difference was found between the hypertensive and the non-hypertensive groups with regard to race (Table 6).

TABLE 6

ADMISSIONS BY SEX AND RACE

Sex	Column I Total Population		Column II Non-Hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
Male	409	(55.4)	372	(56.3)	37	(48.1)
Female	329	(44.6)	289	(43.7)	40	(51.9)
Total Number of Patients	738	(100.0)	661	(100.0)	77	(100.0)
Race						
White	319	(43.2)	278	(42.1)	41	(53.2)
Black	343	(46.5)	312	(47.2)	31	(40.3)
Other	76	(10.3)	71	(10.7)	5	(6.5)
Total Number of Patients	738	(100.0)	661	(100.0)	77	(100.0)

Of the total population, 81.6 percent (602 patients) had burns of 30 percent or less. In the non-hypertensive group, 84.8 percent (561 patients) had burns of 30 percent or less of the total body surface area. In the hypertensive group, 80.6 percent (62 patients) had burns of 50 percent or less of the total body surface area; 53.3 percent (41 patients) of that 80.6 percent had burns of 30 percent or less (Table 7). The highest incidence of hypertension

occurred in the 21-30 percent burn group with 31.2 percent of the hypertensive patients (24 patients) being in this group.

TABLE 7

PATIENTS BY SIZE OF BURN

Size of Burn	Column I Total Population		Column II Non-Hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
20 percent or less	496	(67.2)	479	(72.4)	17	(22.1)
21-30 percent	106	(14.4)	82	(12.4)	24	(31.2)
31-40 percent	51	(6.9)	40	(6.1)	11	(14.3)
41-45 percent	14	(1.9)	13	(2.0)	1	(1.3)
46-50 percent	21	(2.8)	12	(1.8)	9	(11.7)
51-55 percent	9	(1.2)	4	(0.6)	5	(6.5)
56-60 percent	10	(1.4)	7	(1.1)	3	(3.9)
61-65 percent	2	(0.3)	2	(0.3)	0	(0.0)
66-70 percent	9	(1.2)	7	(1.1)	2	(2.6)
71-75 percent	2	(0.3)	0	(0.0)	2	(2.6)
76-80 percent	7	(0.9)	7	(1.1)	0	(0.0)
81-85 percent	2	(0.3)	1	(0.2)	1	(1.3)
86-90 percent	3	(0.4)	3	(0.5)	0	(0.0)
91-95 percent	4	(0.5)	2	(0.3)	2	(2.6)
96-100 percent	2	(0.3)	2	(0.3)	0	(0.0)
Total Number of Patients	738	(100.0)	661	(100.0)	77	(100.0)

The total population was injured most frequently by scald burns (347 patients; 47 percent) or by flame burns (338 patients; 45.8 percent). However, in comparing the non-hypertensive group with the hypertensive group, the difference was statistically significant ($p < .001$). In the non-hypertensive group, 337 patients

(50.9 percent) had scald burns; 10 patients (13 percent) in the hypertensive group had scald burns. In the non-hypertensive group, 274 patients (41.4 percent) had flame burns; 64 patients (83.2 percent) in the hypertensive group had flame burns (Table 8). In adding the number of hypertensive patients with flame burns (64 patients) to the number of hypertensive patients with both flame and electrical burns (2 patients), the hypertensive group consisted of 66 patients or 85.8 percent with flame burns.

TABLE 8

ADMISSIONS BY TYPE OF BURN

Type of Burn	Column I Total Population		Column II Non-Hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
Scald	347	(47)	337	(50.9)	10	(13)
Flame	338	(45.8)	274	(41.4)	64	(83.2)
Electrical	9	(1.2)	8	(1.2)	1	(1.3)
Flame & Electrical	2	(0.3)	0	(0.0)	2	(2.6)
Contact	34	(4.6)	34	(5.1)	0	(0.0)
Chemical	1	(0.1)	1	(0.2)	0	(0.0)
Severe Sunburn	1	(0.1)	1	(0.2)	0	(0.0)
Unknown	6	(0.8)	6	(0.9)	0	(0.0)
Total Number of Patients	738	(100.0)	661	(100.0)	77	(100.0)

There was no difference in the frequency of a family history of high blood pressure or seizures between the non-hypertensive and hypertensive groups nor was there a correlation between these factors and hypertension (Table 9).

TABLE 9

ADMISSIONS BY FAMILY HISTORY OF SEIZURES OR HIGH BLOOD PRESSURE

Family History	Column I Total Population		Column II Non-Hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
Yes - Seizures	7	(0.9)	7	(1.1)	0	(0.0)
Yes - High Blood Pressure	25	(3.4)	22	(3.3)	3	(3.9)
No - Seizures High Blood Pressure	677	(91.7)	610	(92.3)	67	(87.0)
Information Not Available	29	(3.9)	22	(3.3)	7	(9.1)
Total Number of Patients	738	(100.0)	661	(100.0)	77	(100.0)

All patients resuscitated in Parkland Hospital were resuscitated by the Parkland Formula. Errors in resuscitation were associated with underestimation of burn size or failure to deliver the proper amount of calculated fluid due to staff error. Most of the patients who were under-resuscitated were transferred from

other institutions. There was no statistical difference between the non-hypertensive and the hypertensive group in relation to adequacy of resuscitation (Table 10).

TABLE 10

ADEQUACY OF RESUSCITATION

Resuscitation	Column I Total Population		Column II Non-Hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
Adequate Resuscitation	699	(94.7)	628	(95.0)	71	(92.2)
Inadequate Resuscitation	35	(4.7)	29	(4.4)	6	(7.8)
Unable to Determine	4	(0.5)	4	(0.6)	0	(0.0)
Total Number of Patients	738	(100.0)	661	(100.0)	77	(100.0)

The correlation between the use of Phisohex and hypertension is statistically significant ($p < .01$) (Table 11). Of the total population of 738 patients, 503 patients were admitted between June, 1965, and June, 1972. All of these children were routinely washed with Phisohex. The incidence of hypertension in the group bathed with Phisohex was 12.5 percent (63 patients). The incidence of hypertension in the group of 235 patients admitted after the routine use of Phisohex was discontinued in June, 1972, through June, 1975, was 6 percent (14 patients).

TABLE 11

HYPERTENSION ASSOCIATED WITH
THE USE OF PHISOHEX

Phisohex Baths	Column I Total Population	Column II Hypertensive	
	No. of Patients	No. of Patients	Percent of Patients
Yes*	503	63	(12.5)
No**	235	14	(6.0)

*All patients were washed with Phisohex prior to June, 1972; 503 Patients

**No patients were washed with Phisohex from June, 1972-June 1975; 235 Patients

Specifically, the relationship is between the use of Phisohex on patients with flame burns and hypertension ($p < .05$); not between the use of Phisohex on patients with scald or other type of burn injury (Table 12). Of the 77 hypertensive patients, 63 patients (81.8) percent were bathed with Phisohex. Of these 63 patients, 54 had flame burns; 7 patients had scald burns; and 2 patients had other types of burn injury. Fourteen of the 77 hypertensive patients (18.2 percent) did not receive Phisohex baths. Of these 14 patients, 10 patients had flame burns; 3 patients had scald burns; and 1 patient had another type of burn injury.

TABLE 12

RELATIONSHIP BETWEEN USE OF PHISOHEX AND TYPE OF BURN INJURY IN HYPERTENSIVE PATIENTS

Type of Burn Injury	Phisohex	No Phisohex
	No. of Patients	No. of Patients
Flame	54	10
Scald	7	3
Other	2	1
Total	63	14

The mean number of days Phisohex baths were given to the hypertensive group (53.5 days) was twice the number of days the non-hypertensive group was bathed with Phisohex (26.7 days) (Table 13). The development of hypertension occurred 90 percent of the time during the first 14 days postburn but did develop as late as 51 days postburn. There was no difference between the day of onset of hypertension in the Phisohex and non-Phisohex groups.

TABLE 13

NUMBER OF DAYS PHISOHEX BATHS WERE GIVEN

No. of Days of Phisohex Baths	Column I Total Population	Column II Non-Hypertensive	Column III Hypertensive
Range of Days	1 - 24 days	1 - 248 days	6 - 176 days
Mean Day	30	26.7	53.5

The correlation between sepsis and hypertension (Table 14) was statistically significant ($p < .01$). Non-hypertensive patients had a sepsis rate of 5.9 percent (38 patients); hypertensive patients had a sepsis rate of 31.2 percent (24 patients). There was no difference between the mean day of onset of sepsis (postburn day 13) in the hypertensive and the non-hypertensive group (Table 15).

TABLE 14

OCCURRENCE OF SEPSIS

Occurrence of Sepsis	Column I Total Population		Column II Non-Hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
Yes	63	(8.5)	39	(5.9)	24	(31.2)
No	675	(91.5)	622	(94.1)	53	(68.8)
Total Number of Patients	738	(100.0)	661	(100.0)	77	(100.0)

TABLE 15

POSTBURN DAY OF SEPSIS

Post-burn Day Diagnosis of Sepsis	Column I Total Population	Column II Non-Hypertensive	Column III Hypertensive
Range of Days	1 – 58	1 – 58	1 – 27
Mean Day	13.4	13.2	13.7

In an attempt to determine whether or not family structure had an influence on the incidence of hypertension, the nuclear family data was obtained from the charts. There was no difference in the type of family in the hypertensive and non-hypertensive children with regard to family structure. Nor was there any significant difference between the hypertensive and non-hypertensive group in relation to family members injured in the burn-related accident (Table 16) or when the patient was a battered child (Table 17).

TABLE 16

FAMILY MEMBERS INVOLVED IN THE BURN-RELATED ACCIDENT

Family in Accident	Column I Total Population		Column II Non-Hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
Yes	56	(7.6)	47	(7.1)	9	(11.7)
No	682	(92.4)	614	(92.9)	68	(88.3)
Total Number of Patients	738	(100.0)	661	(100.0)	77	(100.0)

TABLE 17

PATIENTS CLASSIFIED AS BATTERED CHILDREN

Battered Child	Column I Total Patients		Column II Non-Hypertensive		Column III Hypertensive	
Yes	24	(3.3)	20	(3.0)	4	(5.2)
	714	(96.7)	641	(97.0)	73	(94.8)
Total	738	(100.0)	661	(100.0)	77	(100.0)

A comparison was made between the hypertensive and the non-hypertensive groups with relation to pulmonary injury. The incidence of pulmonary injury (Table 18) was significantly higher in hypertensive patients as compared to the non-hypertensive group ($p < .007$).

TABLE 18

PATIENTS WITH PULMONARY INJURY

Pulmonary Injury	Column II Non-Hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
Yes	17	(10)	7	(29)
No	644	(90)	70	(71)
Total Number of Patients	661	(100.0)	77	(100.0)

The correlation between death and hypertension (Table 19) was found to be statistically significant ($p < .001$). The hypertensive group had a death rate of 15.6 percent (12 patients) compared to 5.0 percent (33 patients) in the non-hypertensive group.

TABLE 19

SURVIVAL RATES

Survival	Column I Total Population		Column II Non-hypertensive		Column III Hypertensive	
	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients	No. of Patients	Percent of Patients
Yes	693	(93.9)	628	(95.0)	65	(84.4)
No	45	(6.1)	33	(5.0)	12	(15.6)
Total Number of Patients	73	(100.0)	661	(100.0)	77	(100.0)

Analysis of the additional data collected on the hypertensive patients (Appendex D-1, D-2) which included white blood cell counts, hemoglobin, hematocrit, platelets, blood urea nitrogen, creatinine, sodium, potassium, chloride, calcium, sickling trait, diastolic blood pressure, maximum daily temperature, daily intake and output, and daily weights was conducted using Kendall Tau-B correlation coefficients. The results reflected a significant difference from zero, but the difference was not of a magnitude to be clinically significant in determining cause and effect in relation to any single factor studied or to any combination of factors. There was no correlation between recorded values and average blood pressures over a 24 hour period nor between the values and average blood pressures over the length of time of hospitalization. About half of the patients were treated with anti-hypertensive medications.

The development of hypertension occurred 90 percent of the time during the first 14 days postburn but did develop as late as 51 days postburn. All surviving patients were normotensive by the time of discharge.

Summary of Findings

The data is presented with analysis of each of the three populations; total population, non-hypertensive group, and hypertensive group. The incidence of hypertension was 10.4 percent of the total population.

The distribution of patients admitted during the year was uneven with 42.7 percent being admitted in the first 4 months. June was the lightest month with 5.4 percent being admitted over the 10 year period. The number of patients admitted by year reflects a fairly steady increase in the number of patients being admitted each year but with a very low patient census in the first half of 1975. The hypertensive group does not differ significantly from the non-hypertensive group in month of admission. However, the incidence of hypertension in patients by year of admission is markedly higher in the years 1968, 1971, and 1972.

The total population was fairly well distributed by age group with a slightly larger number of total patients in the 13 to 15 month group and in the 19 month to 2 year group. In the hypertensive group, there were no patients in the 13 to 18 month age group. There was no significant difference by sex or race between the hypertensive and the non-hypertensive group.

Of the total population, 81.6 percent had burns of 30 percent or less of the total body surface area. In the hypertensive group, 80.6 percent had burns of 50 percent or less of the total body surface area with 53.3 percent having burns of 30 percent or less. The highest incidence of hypertension occurred in the 21-30 percent burn group with 31.2 percent of the hypertensive patients being in this group.

The total population was injured most frequently by scald burns, 47 percent of the patients, or by flame burns, 45.8 percent of the patients. However, in comparing the non-hypertensive group with the hypertensive group, the difference was statistically s significant ($p < .001$). In the non-hypertensive group, 50.9 percent of the patients had scald burns; 13 percent in the hypertensive group had scald burns. In the non-hypertensive group, 41.4 percent of the patients had flame burns; 83.2 percent of the patients in the hypertensive group had flame burns. In adding the number of hypertensive patients with flame burns to the number of hypertensive patients with both flame and electrical burns, 85.8 percent of the hypertensive patients had flame burns.

There was no difference in the frequency of a family history of high blood pressure or seizures between the non-hypertensive and hypertensive groups nor was there a correlation between these factors and hypertension. There was also no statistical difference between the non-hypertensive and the hypertensive group in relation to adequacy of resuscitation.

The correlation between the use of Phisohex and hypertension was statistically significant ($p < .01$). However, the relationship was between the use of Phisohex on patients with flame burns and hypertension ($p < .05$); not between the use of Phisohex on patients with other types of burn injury. The mean number of days Phisohex

baths were given to the hypertensive group was twice the number of days the non-hypertensive group was bathed with Phisohex. The mean day of onset of hypertension was not different between the Phisohex and the non-Phisohex group.

The correlation between sepsis and hypertension was statistically significant ($p < .01$). Non-hypertensive patients had a sepsis rate of 5.9 percent; hypertensive patients had a sepsis rate of 31.2 percent. There was no difference between the mean day of onset of sepsis (postburn day 13) in the hypertensive and the non-hypertensive group.

There was no difference in the type of family in the hypertensive and non-hypertensive children with regard to family structure. Nor was there any significant difference between the hypertensive and non-hypertensive group in relation to family members injured in the burn-related accident or when the patient was a battered child.

The incidence of pulmonary injury was significantly higher in hypertensive patients as compared to the non-hypertensive group ($p < .007$). The correlation between death and hypertension was also found to be statistically significant ($p < .001$). The hypertensive group had a death rate of 15.6 percent compared to 5.0 percent in the non-hypertensive group.

Analysis of the additional data collected on the hypertensive patients was performed using Kendall Tau-B correlation coefficients.

The results reflected a signficant difference from zero but the difference was not of a magnitude to be clinically significant in determining cause and effect in relation to any single factor studied nor to any combination of factors. There was no correlation between recorded values and average blood pressures over a 24 hour period, nor between the values and average blood pressures over the length of time of hospitalization.

The development of hypertension occurred 90 percent of the time during the first 14 days postburn but did develop as late as 51 days postburn. All surviving patients were normotensive by the time of discharge.

Summary

In this chapter the analysis of data is presented as to reflect frequency and significance. Differences between the hypertensive and the non-hypertensive patients were presented.

CHAPTER V

SUMMARY, CONCLUSIONS, IMPLICATIONS, AND RECOMMENDATIONS

Summary

Each year at least 2 million persons are burned seriously enough to require medical attention or to restrict their activity for a day or more. Burns outnumber all other causes of death during infancy, childhood, and adolescence with the highest incidence occurring in children under 5 years of age. The child is susceptible to all the complications which can occur with any burn injury, but the pediatric burn patient is also at risk to develop hypertension as a complicating factor during the recovery period. Significant arterial hypertension frequently develops in children with burns. Although the incidence of hypertension is recognized, a description of the high-risk population has not been available. In an effort to describe the pediatric burn population with hypertension, this study was undertaken.

Although the exact etiology of the hypertension which develops in children with burn injuries is not known, there are several clinical states which, by themselves, might produce hypertension. These clinical states have been reviewed in Chapter II and the pathophysiology involved has been presented. Included are the nursing implications related to the care of the pediatric burn patient, in general, along with a review of the literature

available on hypertension as a complicating factor in poliomyelitis since the clinical findings in both poliomyelitis patients and pediatric burn patients with hypertension appears to be similar.

The total number of pediatric patients admitted to Parkland Memorial Hospital, Dallas, Texas, with a primary diagnosis of burns from June, 1965, through June, 1975, was 984. Of this number, 738 patients met the criteria of this study. Hypertensive criteria was met by 77 patients. Data was then collected on these patients from their medical records and placed on the computer. Analysis of the data was performed using the chi-square test and Kendall Tau-B correlation coefficients. Analysis was supervised by Dr. Richard Browne.

The data is presented with analysis of each of the three populations: total population, non-hypertensive group, and hypertensive group. The incidence of hypertension was 10.4 percent of the total population.

The distribution of patients admitted during the year was found to be uneven. The number of patients admitted by year reflects a steady increase in the number of patients being admitted each year but with a very low patient census in the first half of 1975. The hypertensive group does not differ significantly from the non-hypertensive group in month of admission. However, the incidence of hypertension in patients by year of admission is markedly higher in the years 1968, 1971, and 1972.

The total population was fairly well distributed by age group. There was no significant difference by sex or race between the hypertensive and the non-hypertensive group.

Of the total population, 81.6 percent had burns of 30 percent or less of the total body surface area. In the hypertensive group, 80.6 percent had burns of 50 percent or less of the total body surface area with 53.3 percent having burns of 30 percent or less. The highest incidence of hypertension occurred in the 21-30 percent burn group with 31.2 percent of the hypertensive patients being in this group.

The total population was injured as frequently by scald burns as by flame burns. However, in comparing the non-hypertensive group with the hypertensive group, a difference of statistical significance ($p < .001$) was found; 85.8 percent of the hypertensive patients had flame burns.

There was no difference in the frequency of a family history of high blood pressure or seizures between the non-hypertensive and hypertensive groups nor was there a correlation between these factors and hypertension. There was also no statistical difference between the non-hypertensive and the hypertensive group in relation to adequacy of resuscitation.

The correlation between the use of Phisohex and hypertension is statistically signficant ($p < .01$). However, the relationship is between the use of Phisohex on patients with flame burns and

hypertension ($p < .05$); not between the use of Phisohex on patients with other types of burn injury. The mean number of days Phisohex baths were given to the hypertensive group was twice the number of days the non-hypertensive group was bathed with Phisohex. All patients admitted to the pediatric burn service were washed with Phisohex prior to June, 1972; none were washed with Phischex after that time.

The correlation between sepsis and hypertension was statistically significant ($p < .01$). There was no difference between the mean day of onset of sepsis in the hypertensive and the non-hypertensive group.

There was no difference in the type of family in the hypertensive and non-hypertensive children with regard to family structure. Nor was there any significant difference between the hypertensive and non-hypertensive group in relation to family members injured in the burn-related accident or when the patient was a battered child.

The incidence of pulmonary injury was significantly higher in hypertensive patients as compared to the non-hypertensive group ($p < .007$). The correlation between death and hypertension was also found to be statistically significant ($p < .001$).

Analysis of the additional data collected on the hypertensive patients was performed using Kendall Tau-B correlation coefficients. The results reflected a signficant difference from zero but the

difference was not of a magnitude to be clinically significant in determining cause and effect in relation to any single factor studied nor to any combination of factors. There was no correlation between recorded values and average blood pressures over a 24 hour period, nor between the values and average blood pressures over the length of time of hospitalization.

The development of hypertension occurred 90 percent of the time during the first 14 days postburn but did develop as late as 51 days postburn. All surviving patients were normotensive by the time of discharge.

Conclusions

According to the findings of this study, the following conclusions are made:

1. The incidence of hypertension is 10.4 percent.
2. The pediatric patient with flame burns is the most likely to develop hypertension at some time during his hospital course.
3. The pediatric patient with flame burns who is bathed with Phisohex is twice as likely to develop hypertension at some time during his hospital course.
4. The death rate among hypertensive children is significantly higher than that of the non-hypertensive group.

5. A correlation does exist between pulmonary injury and the development of hypertension.
6. The sepsis rate among hypertensive children is significantly higher than the sepsis rate in non-hypertensive children.
7. The mean day of onset of sepsis is postburn day 13 for both the hypertensive and the non-hypertensive group.
8. There is no correlation between age, sex, race, inadequate resuscitation, or family composition and the development of hypertension.
9. There is no correlation between any of the laboratory data (Appendix D-1, D-2) and the development of hypertension.
10. There is no correlation between any groupings of laboratory data and the development of hypertension.
11. Hypertension generally develops during the first 14 days postburn but can develop at any time during the hospital course.
12. The hypertension subsides with covering of the burn wound and did not persist beyond discharge in any of the patients in this study.

Implications

Hypertension in the pediatric burn patient has implications for nursing administration, nursing education, and nursing practice. Nursing administrators must be aware of the problem of hypertension and encourage recognition by the nursing staff. They can assure the fact that the understanding is incorporated into staff development programs. In addition, the audit can be utilized as a tool to ascertain whether or not blood pressures have been monitored. Nursing educators must be aware of the problem of hypertension and include the problem in the curriculum. The pediatric burn patient at highest risk for the development of hypertension is one with flame burns. The patient may develop hypertension at any time during hospitalization but 90 percent of the patients with hypertension will have developed it within the first 14 days postburn. Nurses must be especially aware of the high-risk patient and assure that blood pressure readings are taken routinely for the first two weeks after admission and daily until the time of discharge. Many times the taking of blood pressures is not done on patients with burns which involve both upper extremities. The nurse in charge must make the staff aware of methods to monitor blood pressures in these children. One method is to wrap the upper arm with one layer of fine mesh gauze over the topical ointment of choice and place the blood pressure cuff leaving a small open area at the anticubital space for the stethescope. If the blood pressure is not audible with a stethescope, a Doppler may be used with good results. An alternative

method is the use of blood pressure readings taken on the leg, using the popliteal space to monitor blood pressure levels with either a stethescope or a Doppler

The added components of a pulmonary injury increases the chance of any patient developing hypertension. Since the incidence of hypertension in children who are given Phisohex baths is twice that of those patients who are not given Phisohex baths, the nurse must assure that all pediatric burn patients are washed with an agent other than Phisohex. Although the relationship between hypertension and sepsis does exist, its etiology is unclear. Therefore, the nurse must assure that routine blood pressures are taken on any patient with sepsis. And, finally, the nurse must be aware that the mortality rate for children with hypertension is significantly higher than in non-hypertensive children and, therefore, must be prepared to deal with any crisis which may arise. Although, at this point, the hypertension which develops as a result of the burn injury does not appear to be preventable, the nurse must alert the medical staff to the existence of the hypertension so that treatment may be properly and rapidly instituted.

Recommendations

Based on the findings of this study, the following recommendations are made:

1. Further study should be undertaken to evaluate pediatric burn patients in a prospective manner in relation to assessment of the nursing methods

used to obtain blood pressure readings, i.e., size of cuff and anatomical placement of cuff.

2. Further study should be conducted to evaluate pediatric burn patients in a prospective manner in relation to plasma renin and urine catecholamine levels as they relate to hypertension.

3. Further study should be undertaken to evaluate pediatric burn patients in a prospective manner to identify psychological mechanisms adopted in response to the burn injury.

4. Further study should be conducted in a prospective manner to determine the correlation between clinical data and psychological status in relation to hypertension.

5. Further study should be pursued in biomedical laboratories in an attempt to isolate etiologic factors.

6. Further study should be pursued in a prospective manner to determine the correlation between inhalation injury and the development of hypertension.

7. Further study should be conducted in a prospective manner to determine the correlation between hypertension and burn wound sepsis and sepsis from other etiological factors.

8. Further study should be conducted in a prospective manner to determine the correlation between hypertension and pulmonary artery pressures.

APPENDIX A

PERCENTILE VALUES FOR BLOOD PRESSURE BY AGE

AGE	DIASTOLIC PRESSURE 50% + 20
0 to 6 mo	45 / 65
6 mo to 3 yr	64 / 84
4 yr to 5 yr	65 / 85
6 yr to 10 yr	70 / 90
11 yr to 13 yr	70 / 90

SOURCE: Mitchell, S.: Blout, G.G.; Blumenthal, S.; Hoffman, J.I.E.; Jesse, M.J.; Lauer, R.M.; and Weidman, W.H. "The Pediatrician and Hypertension." Pediatrics. Vol. 56(1), July 1975.

APPENDIX B

Normal Laboratory Values

1974-75 Manual for Clinical Pathology Laboratories
Parkland Memorial Hospital

Sodium		135-145 MEq/L
Potassium		3.2-4.5 mEq/L
Chloride		95-105 mEq/L
Calcium (serum)		8.8-10.5 mg%
Blood urea nitrogen		8-22 mg%
Creatinine		0.7-1.3 mg%
Hemoglobin	(Female)	12-16 Gm%
	(Male)	14-18 Gm%
Hematocrit	(Female)	37-47%
	(Male)	42-52%
White blood cell		5,000-10,000/ccm
Platelets		250,000-450,000/ccm

APPENDIX C

DEMOGRAPHIC DATA

CASE NUMBER

NAME

AGE

SEX

RACE

PER CENT OF BURN

TYPE OF BURN

ADMISSION DATE

DID THIS PATIENT DEVELOP HYPERTENSION? YES NO

LOCATION OF BURN
ARM - R L
LEG - R L
HEAD
NECK
CHEST
ABDOMEN
BACK

FAMILY HISTORY OF SEIZURES OR HIGH BLOOD PRESSURE

TOTAL FLUIDS GIVEN DURING FIRST 24 HOURS

REQUIRED FLUIDS PER PARKLAND FORMULA

WAS RESUSCITATION ADEQUATE?

ADMINISTRATION OF PHISOHEX BATHS? YES NO IF YES, HOW MANY DAYS?

A CLINICAL DIAGNOSIS OF SEPSIS? YES NO IF YES, GIVE DATE OF DIAGNOSIS.

DEATH? YES NO

FAMILY SITUATION AS AVAILABLE

CASE NUMBER

PAGE NUMBER

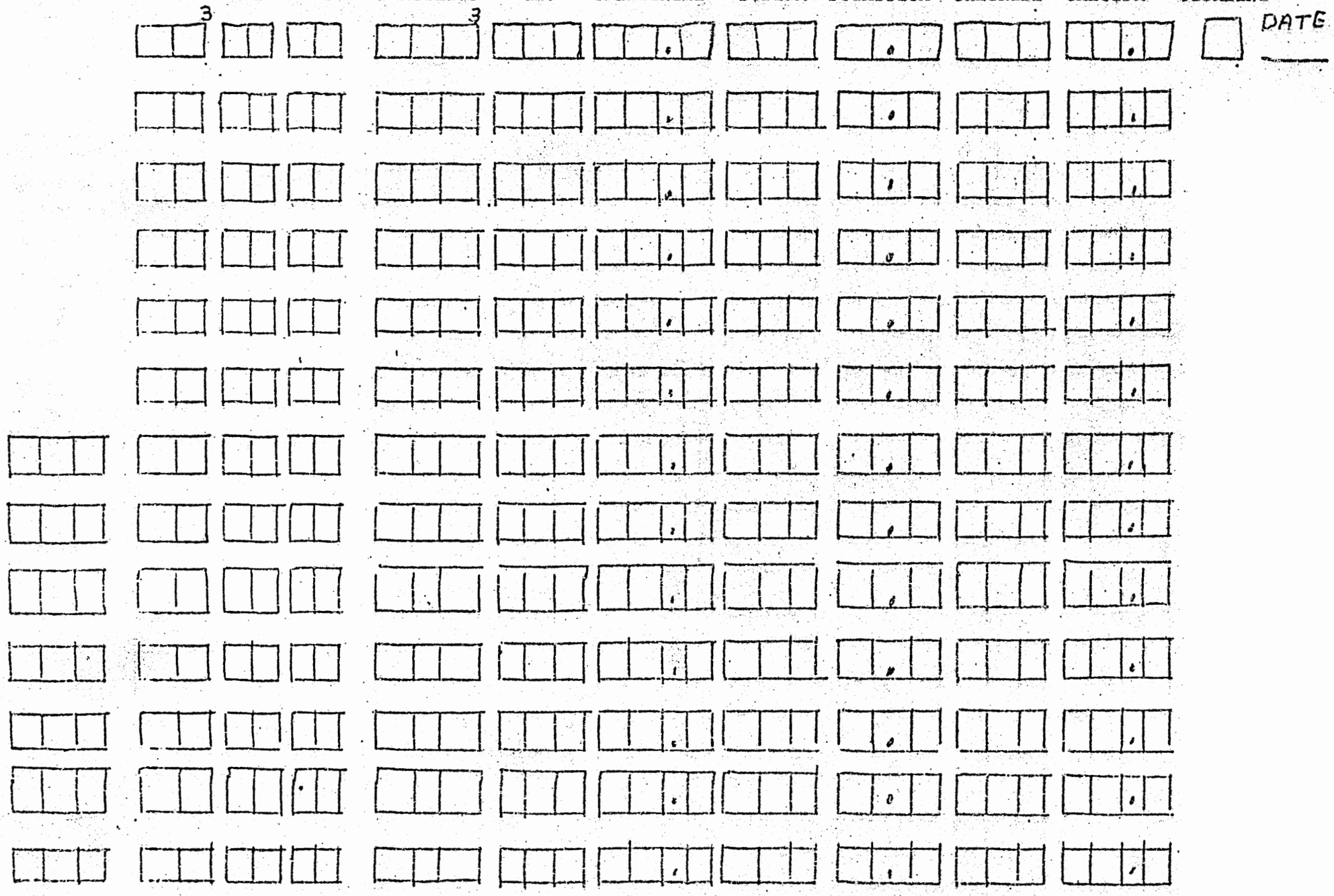
BLOOD PRESSURE, TEMPERATURE, INTAKE AND OUTPUT, WEIGHT

0400	0800	1200	1600	2000	2400	MAX TEMP	TOTAL INTAKE 2	TOTAL OUTPUT 2	WEIGHT KG.

B. P.

CASE NUMBER

WBC, HGB, HCT, PLATELETS, BUN, CREATININE, SODIUM, CHLORIDE, CALCIUM, SICKLING



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AGENCY PERMISSION FOR CONDUCTING STUDY*

THE *PARKLAND MEMORIAL HOSPITAL*

GRANTS TO *MELVA KRAVITZ, R.N.*

a student enrolled in a program of nursing leading to a Master's Degree at Texas Woman's University, the privilege of its facilities in order to study the following problem:

HYPERTENSION IN BURNED CHILDREN

The conditions mutually agreed upon are as follows:

1. The agency (may) (~~may not~~) be identified in the final report.
2. The names of consultative or administrative personnel in the agency (may) (~~may not~~) be identified in the final report.
3. The agency (wants) (~~does not want~~) a conference with the student when the report is completed.
4. The agency is (willing) (~~unwilling~~) to allow the completed report to be circulated thrcugh interlibrary loan.
5. Other: *Forward appendix copy of findings*

Date *10. 7. 75*

Signature of Agency Personnel

Melva Kravitz
Signature of Student

Geraldine Layne RN MS
Signature of Faculty Advisor

*Fill out and sign three copies to be distributed as follows: Original -- Student; first copy -- agency; second copy -- T.W.U. College of Nursing.

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HYPERTENSION IN BURNED CHILDREN

ABSTRACT

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May 1976

This study was undertaken to investigate the incidence of hypertension in the pediatric burn patient. The purposes of the study were to: 1) determine the incidence of hypertension in the pediatric burn population; 2) determine the cause of hypertension in the pediatric burn population; and 3) to describe the pediatric burn population with hypertension.

The population consisted of all patients admitted to the pediatric burn service of Parkland Memorial Hospital, a 797 bed county teaching hospital located in Dallas, Texas, from June 1, 1965 through June 1, 1975. The study was retrospective in nature and conducted by the descriptive non-experimental design to determine if a correlation between hypertension in burn children and significant clinical data could be found. Demographic data (Appendix C) was collected from all charts. Additional clinical data was collected from the charts of all hypertensive patients. Data was analyzed using the chi-square test and Kendall Tau-B correlation coefficients.

According to the findings of this study, the following conclusions were made: 1) the incidence of hypertension was 10.4

percent; 2) the pediatric patient with flame burns was the most likely to develop hypertension at some time during his hospital course; 3) the pediatric patient with flame burns who was bathed with Phisohex was twice as likely to develop hypertension; 4) the death rate among hypertensive children was significantly higher than that of the non-hypertensive group; 5) a correlation did exist between pulmonary injury and the development of hypertension; 6) the sepsis rate among hypertensive children was significantly higher than the sepsis rate in non-hypertensive children; 7) the mean day of onset of sepsis was postburn day 13 for both the hypertensive and the non-hypertensive group; 8) there was no correlation between age, sex, race, inadequate resuscitation, or family composition and the development of hypertension; 9) there was no correlation between any of the laboratory data (Appendix D-1, D-2) and the development of hypertension; 10) there was no correlation between any groupings of laboratory data and the development of hypertension; 11) the hypertension generally developed during the first 14 days postburn but did develop at later times during the hospital course; 12) the hypertension subsided with covering of the burn wound and did not persist beyond discharge in any of the patients studied.