

AD-A267 953 JMENTATION PAGE

Form Approved
OMB No. 0704-0188

is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and reviewing the collection of information, sending comments regarding this burden estimate or any other aspect of this burden to Washington Headquarters Service, Directorate for Information Operations and Reports, 1215 Jefferson Road to the Office of Management and Budget, Paperwork Reduction Project (0704-0188) Washington, DC 20503

1. AGENCY USE ONLY (Leave blank)		2. REPORT DATE MAY 1993		3. REPORT TYPE AND DATES COVERED THESIS/DISSERTATION	
4. TITLE AND SUBTITLE Implications for Advanced Nursing Practice in the Patient with Heat Stress				5. FUNDING NUMBERS	
6. AUTHOR(S) Patricia A Skelton					
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) AFIT Student Attending: Univ of Maryland				8. PERFORMING ORGANIZATION REPORT NUMBER AFIT/CI/CIA- 93-065	
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES) DEPARTMENT OF THE AIR FORCE AFIT/CI 2950 P STREET WRIGHT-PATTERSON AFB OH 45433-7765				10. SPONSORING/MONITORING AGENCY REPORT NUMBER	
11. SUPPLEMENTARY NOTES					
12a. DISTRIBUTION/AVAILABILITY STATEMENT Approved for Public Release IAW 190-1 Distribution Unlimited MICHAEL M. BRICKER, SMSgt, USAF Chief Administration				12b. DISTRIBUTION CODE	
13. ABSTRACT (Maximum 200 words) DTIC ELECT AUG 17 1993 S B D 93-18999 03 8 18 068					
14. SUBJECT TERMS				15. NUMBER OF PAGES 70	
				16. PRICE CODE	
17. SECURITY CLASSIFICATION OF REPORT		18. SECURITY CLASSIFICATION OF THIS PAGE		19. SECURITY CLASSIFICATION OF ABSTRACT	
				20. LIMITATION OF ABSTRACT	

Implications for Advanced Nursing Practice
in the Patient with Heat Stress

by

Patricia A. Skelton

DTIC QUALITY INSPECTED 3

Seminar paper submitted to the Faculty of the Graduate School
of the University of Maryland in partial fulfillment
of the requirements for the degree of
Master of Science
1993

Accession For	
NTIS GRA&I	☒
DTIC TAB	☐
Unannounced	☐
Justification	
By	
Distribution/	
Availability Codes	
Dist	Avail and/or Special
A-1	

APPROVAL SHEET

Title of Seminar Paper: Implications for Advanced
Nursing Practice in the
Patient with Heat Stress

Name of Candidate: Patricia A. Skelton
Masters of Science, 1993

Seminar Paper and Abstract Approved by:

Marguerite Littleton Kearney
Marguerite Littleton Kearney, DNSc, RN
Associate Professor
Graduate Program in Trauma/Critical
Care Nursing

Dorrie Fontaine
Dorrie Fontaine, DNSc, RN
Associate Professor
Graduate Program in Trauma/Critical
Care Nursing

Date Approved:

10 May 93

Abstract

Heat stress is non-discriminating. Under the right conditions, it can affect anyone. The intent of this paper is to explore the nature of heat-related injuries, current management techniques, and prevention methods. Because heat stress, at its extreme, can pose a life-threatening medical emergency, prompt recognition and initiation of therapy is vital in minimizing adverse effects and potential death. Understanding the physiological basis of thermoregulation in the body is essential. Clinical manifestations and treatment modalities of five heat-related disorders will be reviewed from currently available literature. The value of prevention is also discussed. Concluding remarks present implications for practice within the scope of the Clinical Nurse Specialist role.

Table of Contents

CHAPTER 1:	Introduction.....	1
	History.....	1
	Demographics.....	2
	Risk Factors.....	5
	Social.....	5
	Physiological.....	7
CHAPTER 2:	Physiological Responses to Heat Stress...	12
	Thermoregulation in the Body.....	12
	Heat-Related Disorders.....	18
	Heat Edema.....	18
	Heat Syncope.....	18
	Heat Cramps.....	20
	Heat Exhaustion.....	21
	Heatstroke.....	22
CHAPTER 3:	Assessment and Treatment Modalities.....	27
	Heat-Related Disorders.....	27
	Heat Edema.....	27
	Heat Syncope.....	27
	Heat Cramps.....	28
	Heat Exhaustion.....	29
	Heatstroke.....	31
	Assessment.....	31

	Treatment.....	36
	Supportive Care.....	42
CHAPTER 5:	Prevention.....	47
CHAPTER 6:	The Role of the Clinical Nurse	
	Specialist.....	54
	Expert Practitioner.....	54
	Educator.....	55
	Consultant.....	57
	Researcher.....	59
	Manager.....	60
References.....		63

CHAPTER ONE

Introduction

Heat stress is a non-discriminating health problem. Under the right set of circumstances, it can afflict anyone, at any time. Extreme cases of heat stress pose a life-threatening medical emergency. Heatstroke, the most severe form of heat stress, is responsible for the death of 4,000 people annually in the United States, 20% of whom are young athletes (Carney, 1989). Adverse outcomes can be prevented through prompt recognition and initiation of therapy.

There exists a variety of heat-related disorders. Each has unique physiological responses, assessment parameters, clinical manifestations, treatment methods, and prevention techniques. The Clinical Nurse Specialist (CNS) as expert practitioner, educator, consultant, researcher, and manager, can integrate these roles in managing patients with heat stress.

History

The effects of heat stress have plagued mankind since biblical times (Posey & Caruso, 1986). In 24 B.C., the Roman army was ravaged by the effects of heat in Arabia (Yarbrough, 1991).

Much has been learned in the past 60 years about

the physiology of heat regulation, and subsequently applied to the treatment and prevention of heat stress. Cummings (1983) notes that research findings from the early 1930's acknowledged that vigorous exercise increased body temperature to 101 to 102 degrees Fahrenheit. This was interpreted by researchers at that time as a failure of the autonomic nervous system to thermoregulate (Cummings). In 1945, this rationale was reassessed by Asmussen and Boge (cited in Cummings, 1983) who determined the rise in body temperature was actually due to increased muscle temperature associated with vigorous activity. They found that the increased muscle temperature also raised blood temperature by returning blood from the periphery and muscles to the heart, and ultimately elevated the core body temperature (Cummings). In contrast to earlier findings, they concluded this to be a normal physiologic response.

Demographics

Regarding the incidence of heat-related illnesses, Anderson, Reed, and Knochel (1983) point out that civilians have not fared as well as the military population, who are generally younger, healthier and in better-condition. The last 20 years has seen a surge

in the public's desire for physical fitness, putting more and more people at risk for heat stress. The world of competitive sports, both professional and amateur, has long been recognized as a high risk arena for heat-related illness. The combination of heat waves and high humidity increase this risk.

Only central nervous system injuries and heart failure surpass heatstroke as a cause of death in trained athletes (Kunkel, 1986; Yarbrough, 1991). This is true for high school athletes as well, where there is an average of 10 deaths per year (Cummings, 1983). Furthermore, heatstroke has a mortality rate of 10% to 80% (Carney, 1989). Consequently, the key for health care providers lies in timely and effective intervention.

Heat stress is a constant concern for military service men and women because members participate in rigorous training exercises and must be ready for worldwide deployment at any time. Kunkel (1986) addresses a study of heat disorders in recruits at a southern marine base, which involved cases during the months from April through August. Seventy-three percent of those affected with heat disorders were from northern states. Sixty percent of the sample developed

injuries during the early phases of training, and 93% of injuries resulted either during or immediately after heavy physical exercise. This information is valuable to commanders and instructors who must consider acclimatization of recruits when scheduling training activities. Furthermore, educational programs on prevention should be integrated into the indoctrination to military life.

Kunkel (1986) relates an incident of heat illness in Israeli soldiers after a four kilometer night march, where the temperature was only 78 degrees Fahrenheit! This emphasizes that high temperatures are not necessary to put an individual at risk.

While heat-related disorders are expected in hot climates, they have become more of a problem for inner city and urban dwellers (Posey & Caruso, 1986). More than 1200 reports of heat stress occurred during the heat wave of 1980 (Hayes, 1984). "The urban environment intensifies the effects of elevated temperatures and humidity due to the trapping of hot air in the poorly ventilated stagnant atmosphere and reflective nature of concrete areas" (Posey & Caruso, 1986, p. 216).

There is a limit to the amount of heat the body

will tolerate. Heightened awareness and understanding is vital, not only for professional athletes, soldiers, and workers, but for the general public as well, especially those with increased risk factors. The ability to immediately recognize and accurately diagnose the ill effects of heat stress is imperative to minimize and/or avoid adverse consequences.

Risk Factors

Many factors exist that place an individual at risk for this potentially life-threatening medical emergency. Who is at risk? Age, state of health, level of activity, and environmental factors play important roles in response to heat stress (Birrer, 1988). Posey and Caruso (1986) present two distinct categories in the identification of high risk individuals: those with social factors and those with physiological factors.

Social risk factors include: advanced age, low income, obesity, psychiatric disorders and alcoholism (Posey & Caruso, 1986). Physiological factors include those suffering acute infections, cardiovascular disease, impaired sweat production, and endocrine disorders. The use of certain drugs, such as amphetamines and diuretics, also presents a

physiological risk (Posey & Caruso). A brief review of these factors as implicated in heat stress follows.

Social

The elderly population has a poor tolerance to high temperatures (Walker, 1986). They sweat less, have slower circulation, and a decreased cardiovascular response (Hayes, 1984). All of these contribute to susceptibility. Data collected during the summer of 1984 in New York City indicates elderly women have a higher mortality rate associated with a "sudden and severe heat wave" (Centers for Disease Control, 1984, p. 430).

The very young, like the very old, respond more slowly to temperature changes (Birrer, 1988). Because infants lack insulating fat and do not produce adequate sweat, they have no protection and are unable to cool themselves through evaporative cooling mechanisms (Hayes, 1984). Young children have increased metabolic heat production due to high activity levels and fewer sweat glands. Consequently, they experience greater problems with heat dissipation (Birrer).

Poverty and low income status impact ones exposure to extreme heat. The urban poor live in cramped housing conditions with poor ventilation and

insulation. Hot air becomes trapped and ambient temperatures soar. Little or no access to swimming pools, the inability to afford air conditioning or fans, as well as the lack of education regarding heat stress prevention, all contribute to increased heat-related disorders (Cummings, 1983; Hayes, 1984).

Obese individuals have problems dissipating heat because of lower surface area to weight ratios, lower specific body heat, and a generally poor level of cardiovascular fitness. Fat, in and of itself, prevents heat from escaping (Walker, 1986). Body build also affects heat production in stocky individuals with thick muscle mass, because more muscle mass means more metabolic activity (Birrer, 1988).

The severe agitation observed in psychiatric disorders, delirium tremens, or seizures, increases muscle activity, which subsequently increases body temperature (Cummings, 1983). When combined with pre-existing heat exposure, the cumulative effect of heat gain poses a serious threat to individuals in agitated states. Alcohol intake should be avoided during episodes of heat exposure. It impairs judgement, increases metabolic heat load, contributes to dehydration through suppression of antidiuretic

hormone, and causes peripheral vasodilation (Barrett, 1991; Walker, 1986). These factors facilitate heat gain by adding to the elevated heat load and interfering with heat dissipation.

Physiological

Physiological factors also impact an individual's response to heat stress. Prior heat illness places an individual at increased risk for future episodes of heat-related disorders, due to damage of the regulatory mechanism (Posey & Caruso, 1986, Walker, 1986).

Infection or recent immunization increases the risk of heat-related disorders, especially when associated with the increased metabolic activity and heat production of strenuous exercise (Cummings, 1983). Fever from infection or immunization reactions exacerbates the effects of heat stress, amplifying the amount of heat the body is attempting to dissipate (Birrer, 1988).

Cummings (1983) relates an example where military recruits with mild shigellosis, developed heatstroke while on maneuvers in the desert.

The efficiency of the heart to pump blood to muscles, skin, and vital organs plays an important role in heat loss. Consequently, for those suffering from cardiovascular disease, heat stress represents a

serious threat. Heart disease interferes with the efficiency of the cardiovascular system to respond appropriately. Diminished pumping ability reduces stroke volume, cardiac output and perfusion. This decreased ability to maintain adequate blood flow to peripheral blood vessels impairs heat loss (Walker, 1986).

Impaired sweat production due to skin diseases, such as ectodermal dysplasia (no sweat glands), or severe burns, where sweat glands are lost, affects the body's natural cooling mechanisms (Cummings, 1983). Loss of sweat production capability prevents cooling through evaporation. As a result, these individuals are at increased risk for heat stress. Abnormal skin conditions, such as scleroderma and scarring, reduce the number of sweat glands, which subsequently interferes with heat dissipation through evaporation (Birrer, 1988).

Endocrine disorders alter heat regulation in the body through several mechanisms (Hayes, 1984). In cystic fibrosis, abnormal sweat production and composition interfere with sweating and evaporative cooling potential. Diabetic patients are at increased risk for circulatory disorders and neuropathies, which

can interfere with peripheral vasodilation.

Hyperthyroid conditions increase metabolic activity and therefore heat production. This additional heat load can certainly aggravate the condition of a patient experiencing heat stress.

A wide variety of medications and drugs interfere with the body's heat regulating efforts. Their actions either increase metabolic activity and heat production, interfere with the ability to either sweat or peripherally vasodilate, or cause dehydration (Walker, 1986). Amphetamines, increased doses of thyroid medication, and street drugs, such as lysergic acid diethylamide (LSD), phencyclidine hydrochloride (PCP), and cocaine, increase metabolic activity and heat production (Birrer, 1988; Cummings, 1983; Knochel, 1989). Hyperthermia from convulsions in cocaine poisoning can be life-threatening (Knochel). Phenothiazines, antihistamines, and anticholinergics inhibit sweating (Carney, 1989; Cummings, 1983). Antiparkinsonians, antipsychotics, tricyclic antidepressants, and belladonna synthetic alkaloids are also included in this category. Diuretics, alcohol, and excessive use of laxatives cause dehydration, which impairs heat regulation (Birrer, 1988; Carney, 1989).

Determination of the use of prescription, over-the-counter, and/or illegal drugs is an essential part of patient assessment. Many people do not read inserts that accompany medication and are unaware of potential actions and side effects. This is particularly true with over-the-counter medications, such as antihistamines, diuretics, cough preparations with alcohol, and laxatives (Barrett, 1991). Because medications can seriously interfere with the body's thermoregulatory efforts, a high index of suspicion is necessary in assessing heat-related disorders to appropriately diagnosis and treat heat stress victims.

Summary

Awareness of risk factors and the role they play in the development of heat stress disorders can greatly influence potential outcomes of heat-related injuries. A reliable medical history along with relevant environmental conditions will assist health care professionals in quickly and accurately assessing the true nature of the patient's condition. This information will prevent delays in treatment and improve the victim's chance for uncomplicated recovery.

CHAPTER TWO

Physiological Responses to Heat Stress

Temperature Regulation

Nervous feedback mechanisms, operating through the temperature-regulating centers in the hypothalamus, are responsible for thermal regulation in the body (Guyton, 1991). The function of this system is to maintain a constant temperature to allow peak efficiency of all cells in the body (Walker, 1988). The range of normal body temperatures is 97 to 99.5 degrees Fahrenheit or 36 to 37.5 degrees Celsius (Guyton).

When heat production or gain in the body exceeds heat loss, temperature increases. Heat acquisition occurs through a variety of mechanisms. Internal heat production is a by-product of basal metabolic processes of vital organs, such as the liver, brain, heart, and skeletal muscle (Guyton, 1991). Walker (1988) cites two other sources of heat gain in the body: externally from the environment and that which is internally generated from muscle activity. Environmental heat gain may result from exposure to large doses of direct sunlight, being enclosed in hot vehicles or buildings with poor or no ventilation, and saunas or hot tubs (Carney, 1989). Heat production in the body can

increase 12 to 15 times normal during periods of hard, physical labor or vigorous exercise (Carney, 1989). Muscle activity from seizures and shivering can also increase heat production.

Heat receptors in the skin and deep tissues of the body stimulate the posterior hypothalamus to regulate body temperature (Guyton, 1991; Walker, 1988). Heat sensitive neurons in the hypothalamus, responding to increased body temperature, initiate cardiovascular and sweat reflexes (Guyton, 1991). These reflexes cause sweating and peripheral vasodilation, protective internal mechanisms that return the rising temperature to normal.

"The integrity of the cardiovascular system is the key to effective thermoregulation" (Anderson et al., 1983, p. 117). Vasodilation allows body heat to escape by maximizing the body's cooling surface area. Heat is transferred from internal organs to the skin via the circulatory system, where it is lost to the surrounding environment (Guyton, 1991; Walker, 1988). The highly vascular nature of subcutaneous tissue enables accommodation of up to 30% of cardiac output (Guyton). The rate of flow is controlled by the sympathetic nervous system through vasodilation or vasoconstriction

of arterioles and arterio-venous anastomoses which respond to body and environmental temperature changes (Guyton). This ability to increase blood flow and dilate blood vessels in skin is crucial to heat loss (Anderson et al., 1983).

Heat is eliminated from the body through four mechanisms: radiation, conduction, convection, and evaporation. The first three, however, are only effective in cooling when air temperature is less than skin temperature, usually around 92 degrees Fahrenheit (Cummings 1983). Evaporation is the only mechanism effective when ambient temperatures are greater than 92 degrees Fahrenheit (Cummings).

Radiation, defined as the loss of heat from the skin through electromagnetic waves, can account for up to 60% of heat loss from the body (Carney, 1989; Vander, 1990). It is important to remember that radiation can also be a source of heat gain in warm environments (Yarbrough, 1991).

Conduction occurs with the transfer of heat from warmer to cooler surfaces through direct contact (Cummings, 1983). Loss of heat by direct conduction is minimal, estimated by Guyton (1991) at three percent. while loss by conduction to air is significantly

greater at 15% (Guyton). Heat gain will occur if direct contact is with a source of greater heat (Cummings).

An additional 12 to 15% of heat loss can be attributed to convection, the loss of heat through movement of air or water next to the body (Carney, 1989). Once air or water next to the skin is the same temperature as the skin, there is no further heat loss; and as a consequence, only movement will bring cooler air next to the skin (Guyton, 1991). While air is a poor conductor of heat, water has excellent thermal conductivity at 32 times greater (Guyton). This ability to absorb larger amounts of heat makes water immersion an extremely effective cooling mechanism.

Vander (1990) defines evaporation, also called vaporization, as the transformation of water from a liquid to gas state. This process can remove heat faster than 10 times the normal basal rate of heat production (Guyton, 1991). Thus, evaporation is also a very effective cooling mechanism.

Evaporative heat loss, however, depends on the ability to generate sweat (Caldroney, 1986). Regulated by the autonomic nervous system, sweating is initiated through stimulation of the hypothalamus in response to

an increased heat load. Sweat glands can also be stimulated by epinephrine and norepinephrine, released during exercise (Guyton, 1991). There is a limit, however, to the amount of vaporization and therefore, how much heat can be eliminated by the body (Cummings, 1983).

A factor critical to the body's ability to lose heat through evaporative sweat loss is the amount of relative humidity in the air (Cummings, 1983; Kunkel, 1986; Vander, 1990). The greater the humidity, the less capable the body is of losing heat (Kunkel, 1986). When ambient air becomes saturated with moisture, sweat becomes a blanket on the skin that retains body heat and increases body core temperature (Carney, 1989).

With increased temperature, the body gains heat, and with elevated humidity, evaporation slows (Hayes). Conditions where temperatures are greater than 95 degrees Fahrenheit (35 degrees Celsius) and humidity exceeds 75%, inhibit the evaporative process that is necessary for cooling and multiplies the risk of heat illness (Birrer, 1988; Hayes).

High temperatures, however, are not required to find oneself in trouble, especially with vigorous activity and high humidity. A temperature of 90

degrees Fahrenheit with 40% humidity is just as dangerous as a temperature of 70 degrees Fahrenheit with 80% humidity (Birrer). When ambient temperature and humidity stay below these values, the individual is able to cool off through evaporative sweating. However, if these values are exceeded, cooling becomes less effective.

A heat index, also known as a wet-bulb globe temperature, is a far more accurate indicator of the "real" amount of environmental heat (Birrer, 1988; Jacobson, 1992). This measurement reflects the combined effects of ambient temperature, relative humidity, radiant heat, and wind velocity (Jacobson, 1992). Based on these factors, the level of risk for heat illness is considered low when the heat index is less than 65 degrees Fahrenheit, moderate at 65 to 73 degrees Fahrenheit, and high when it is above 73 degrees Fahrenheit (Birrer).

Vander (1990) defines acclimatization as "environmentally induced improvement in functioning of a genetically based physiological system" (p. A-14). According to Dr. Robert J. Stine (Kunkel, 1986), several adaptive processes take place with acclimatization: an improved metabolic efficiency

results in increased aerobic metabolism; sweating is promoted earlier and at lower body temperatures; and cardiac output and stroke volume increase, while peak heart rate decreases, resulting in increased heat loss.

With acclimatization, sweat composition changes (Jacobson, 1992). A decrease in plasma volume from hypovolemia triggers the secretion of aldosterone, which conserves sodium in the body. As a result, sweat becomes less hypotonic (Anderson, et al., 1983; Kunkel, 1986).

Adaptation also involves changes in the volume of sweat. The unacclimated individual sweats at a maximum rate of 1.5 liters/hour (Jacobson, 1992). However, when exposed to hot weather for one to six weeks, this amount can reach two to three liters/hour (Guyton, 1991; Kunkel, 1986). The process may take 10 to 20 days, with full tolerance not being achieved for up to two months of exposure (Kunkel).

Heat Related Disorders

Caldronney (1986) identifies five heat-related disorders: heat edema, heat syncope, heat cramps, heat exhaustion, and heat stroke. He maintains that the first three are essentially benign conditions, yet are potential precursors to more extreme forms of heat

stress.

Heat Edema

Although the exact cause of heat edema is unclear, it is believed to be caused by a "relative hyperaldosteronism" brought on by heat exposure (Kunkel (1986). Abnormal fluid retention is thought to result from cutaneous dilation of blood vessels and/or an increase in antidiuretic hormone secreted by the hypothalamus (Parks & Calabro, 1990). In response to heat stress and the loss of fluids, the body attempts to preserve sodium and water to maintain its overall integrity. Heat edema is usually limited to dependant areas, such as hands and lower extremities (Kunkel).

This condition is more prevalent in older and unacclimated individuals (Kunkel, 1986). A generally slower cardiovascular response in older individuals interferes with vasodilation for heat elimination. In addition, the unacclimated individual loses a lot of sodium in sweat. This prompts increased aldosterone secretion to preserve sodium, which pulls fluid into extracellular spaces, resulting in edema (Anderson, et al., 1983).

Heat Syncope

Zal (1984) refers to heat syncope as a minor

disorder. It is a "condition thought to occur as a result of an increased blood plasma volume and cutaneous vasodilation in response to heat exposure" (Parks & Calabro, 1990, p. 27). Kunkel (1986) explains that peripheral vasodilation, the result of increased body temperature and sympathetic nervous system response, alters hemodynamics and causes inadequate venous return to vital organs. In this case, symptoms are observed when the brain is affected by altered circulation. Volume depletion from large amounts of sweating contributes to decreased venous return (Kunkel). The unacclimated individual can lose up to 1.5 liters of sweat per hour when exposed to high temperature and humidity. Therefore, intravascular volumes are depleted from excessive sweat loss, which reduces venous return and filling pressures. A reduced stroke volume and cardiac output lead to decreased tissue and organ perfusion.

Heat Cramps

Though not considered dangerous (Hayes, 1984), heat cramps usually afflict younger, well-acclimated individuals, such as athletes and industrial workers. These people can produce large amounts of dilute sweat (Kunkel, 1986; Zal, 1984). Heat cramps are due to

sodium deficiency incurred as a result of profuse sweating (Birrer, 1988; Hayes, 1984). Cramping affects those who replace the fluids lost with water, and neglect salt losses (Knochel, 1989; Yarbrough, 1991). Despite lower sweat sodium concentrations in acclimated individuals, their total sodium loss may still be higher than that of the unacclimated individual (Anderson, et al., 1983).

Painful spasms occur in large skeletal muscles, usually the arms and legs, during or following heavy work or strenuous physical activity (Yarbrough, 1991; Zal, 1984). They have also been reported to occasionally affect muscles in the abdomen and back (Barrett, 1991). Duration can be brief, lasting less than a minute, or persist for several minutes (Barrett, 1991).

Heat Exhaustion

Heat exhaustion, or heat prostration, is considered by Birrer (1988) as "the most serious heat-related illness in which thermoregulation is still maintained" (p. 10-11). Yarbrough (1991) adds that it is the most common clinical syndrome observed in those working in a hot environment. It often precedes heat stroke and is frequently associated with poor

acclimatization, where cardiovascular, endocrine, and exocrine adaptation has not yet occurred (Anderson, et al., 1983; Price, 1984).

There are two distinct forms of heat exhaustion: hypertonic (water depletion) and hypotonic (salt depletion). However, most cases of heat exhaustion are mixed.

Caldronney (1986) notes that hypertonic heat exhaustion is commonly observed in otherwise healthy individuals, and results from excessive physical exertion in hot, humid weather. He defines this as true dehydration--large losses of hypotonic sweat and limited fluid intake. Circulating blood volume is depleted and creates a strain on competing demands for blood flow to skin and active muscles (Barrett, 1991).

On the other hand, hypotonic heat exhaustion, is seen more often in sedentary and elderly individuals exposed to high temperatures (Caldronney, 1986). Sweat loss, which depletes the body of water and sodium, is only replaced with large amounts of water, not sodium (Caldronney). The negative sodium balance that results usually occurs over several days (Jacobson, 1992).

Although less critical than heat stroke, heat exhaustion demands appropriate and timely intervention

(Hayes, 1984). The effects of prolonged heat exposure are cumulative, and if left untreated can progress to heat stroke, a life-threatening condition.

Heatstroke

Heatstroke, the most severe form of heat-related injuries, is the result of a "profound dysfunction of the body's heat-regulating mechanism" (Carney, 1989, p. 25). Early symptoms of heatstroke include respiratory alkalosis, dehydration, deranged electrolytes, acidosis, and a low glucose. Later symptoms, eight to 24 hours after the initial insult, are sequelae to the direct effect of heat, such as cell damage and electrolyte shifts. Arrhythmias are usually associated with electrolyte imbalances and cardiac injury. Stress from the increased demand placed on the cardiovascular system to accommodate heat loss causes tachycardia, increased cardiac output and raises the potential for myocardial infarctions (Posey & Caruso, 1986).

Birrer (1988) divides heatstroke into two categories: classic and exertional. Passively acquired, classic heatstroke affects those at the extremes of age, the sedentary, and individuals with chronic illnesses. In contrast to exertional heatstroke, these patients usually have some

predisposing medical illness (Jacobson, 1992). For instance, cardiopulmonary patients have limited ability to perfuse peripherally. Diabetics are often afflicted with cardiovascular disease and peripheral neuropathy, while conditions of abnormal skin or sweat glands interfere with sweating and evaporative cooling. In heatstroke, complications of pre-existing medical illnesses manifest as sepsis, pulmonary edema, gastrointestinal bleeding and/or cardiac pathology (Jacobson). With a mortality rate of up to 80%, this classic type of heatstroke is frequently observed during heat waves (Birrer, 1988; Reed & Anderson, 1986).

Victims typically present with hot, dry skin (Roberts, 1992). Loss of sweating capability is associated with failure of heat-regulating mechanisms and/or lack of sweat glands. Because of a slow, insidious onset, patients will have only a moderately decreased potassium and mild respiratory alkalosis. Lactic acidosis is uncommon and represents a more ominous outcome (Reed & Anderson, 1986).

Exertional heatstroke is primarily observed in young, healthy individuals, and carries a 20% mortality (Birrer, 1988). Disorders associated with

exertional heatstroke include lactic acidosis, renal failure, and disseminated intravascular coagulation (Reed & Anderson, 1986). Rhabdomyolysis, the breakdown of muscle tissue, is frequently observed in this type of heatstroke. As muscle tissue is destroyed, myoglobin is released along with potassium. Myoglobin can clog renal tubules, slowing or obliterating filtration. These factors pose serious problems for the heatstroke patient whose kidneys may fail from hypotension and poor perfusion, and are unable to filter and excrete appropriately.

People with this type of heatstroke are usually still wet and sweating (Roberts, 1992). Exertional heatstroke is a more acute process and reflects a less profound dehydration. Yarbrough (1991) corroborates this, finding that sweating can persist in up to 50% of cases.

When body temperatures exceed 105 degrees Fahrenheit, parenchymal damage occurs in all cells (Posey & Caruso, 1986). The result is a disorder which affects every organ system in the body. Caldrony (1986) reports that autopsy results reveal "widespread tissue necrosis and hemorrhage... protein within the vascular endothelium is literally fried" from direct

thermal injury (p. 48N). Heat-related damage to body systems can be attributed to four mechanisms: 1) direct thermal injury to tissues, 2) anoxic and ischemic effect of shock on tissues, 3) homeostasis disruption, and 4) complications from damaged structures and function of tissues (Posey & Caruso, 1986).

Homeostasis is disrupted as metabolism increases from heat and/or exercise. Patients can move into a shock state from poor perfusion and oxygen debt from their hypermetabolic states. This potentiates pump failure, pulmonary edema, and arrhythmias (Kunkel, 1986). A metabolic acidosis results from increased lactic acid production due to anaerobic metabolism, as a result of hypotension, hypovolemia, and increased metabolic demand (Posey & Caruso, 1986). The body attempts to cool itself and eliminate acids by hyperventilating, with respiratory rates often exceeding 60 breaths/minute.

Myocardial failure can also occur secondary to acidosis and hypoxemia. This can be exacerbated by large amounts of intravenous fluid (Reed & Anderson, 1986). While patients with exertional heatstroke may require large amounts of intravenous fluid replacement.

those suffering from classic heatstroke generally do not. Fluid overload may further compromise cardiovascular integrity by increasing filling pressures and oxygen demand.

Summary

While differences in individual response and tolerance capabilities are recognized, the amount of damage ultimately depends on the maximum temperature reached and the duration of time spent at that temperature (Carney, 1989). Death can occur from complications due to damage of the brain, liver, kidney, heart and lungs (Hayes, 1984; Knochel, 1989).

CHAPTER THREE

Assessment and Treatment Modalities

Heat Edema

Though uncomfortable for some, heat edema represents no serious medical danger to otherwise healthy individuals. Nevertheless, appropriate recognition and accurate assessment of heat stress at this early stage is important because of its cumulative effects and potential for progression to more serious forms of heat disorders. Because the victim's volume status is still adequate at this point, vital signs are usually normal (Parks & Calabro, 1990). Symptoms are seen as edema, or swelling, of dependent extremities.

Treatment is supportive in nature, aimed at moving the patient out of the heat and elevating affected extremities (Kunkel, 1986). Explanations by the health care provider are also indicated as to the cause of heat edema and prevention techniques for the future.

Heat Syncope

Characterized by dizziness and sudden, brief fainting episodes, heat syncope usually occurs in the unacclimated individual during extreme heat exposure (Kunkel, 1986). Generally, vital signs and temperature are normal (Kunkel). Hypotension associated with

sudden changes in posture indicates temporary intravascular volume depletion from sweating and maldistribution of blood from peripheral vasodilation.

Transferring the patient to a cooler environment and elevating the lower extremities usually provides adequate relief and resolution of the syncopal episode (Zal, 1984). Fluid replacement may be indicated if hypotension persists in order to restore intravascular volume and improve perfusion (Kunkel, 1986).

Heat Cramps

In contrast to patients experiencing heat syncope, heat cramps are observed more frequently in young, well-acclimated individuals, capable of producing large amounts of dilute sweat. Patients are alert, but may be tachycardic (Carney, 1989). To compensate for the volume depletion from sweating, the heart is stimulated to increase its rate and subsequent cardiac output. Patient temperatures are usually normal (Birrer 1988; Carney, 1989; Knochel, 1989). Serum sodium levels may be low or normal (Hayes, 1984; Kunkel, 1986). Other symptoms include muscle twitching, nausea, weakness, feeling faint, and cool moist skin (Birrer).

Therapy is aimed at replacement of sodium losses after moving the patient to a cool environment (Hayes.

1983). While the tendency may be to massage a muscle cramp, stretching is much more effective (Knochel, 1989). Muscles may be sore and stiff for several days after the incident, but analgesics will not ease the discomfort (Hayes, 1983). Bedrest provides the appropriate muscle rest necessary for improvement.

Patients should be encouraged to eat salty foods, get bedrest for a day or two, and drink plenty of fluids (Hayes, 1984). Oral salt solutions (one teaspoon of salt per eight ounces of water) or "Gatorade" may be used to replace sodium losses if patients are able to tolerate oral fluids (Carney, 1989). Sweetened fluids should be avoided because glucose slows both gastric emptying and absorption (Jacobson, 1992). Birrer (1988) recommends that patients avoid taking oral salt tablets since they are "poorly tolerated and delay gastric emptying, causing stomach irritation, nausea and vomiting" (p. 10). Intravenous fluid replacement may be a better treatment choice, since many patients are nauseated and unable to tolerate fluids by mouth (Carney, 1989). A definitive diagnosis is usually made when the patient responds well to intravenous saline within several minutes to one hour after administration (Knochel, 1989).

Heat Exhaustion

Because of the variety of responses and presentations, heat exhaustion has been mistaken for a flu syndrome (Caldroney, 1986; Knochel, 1989). These "flu-like" symptoms include fatigue, cramps, weakness, headache, dizziness, fainting, and nausea/vomiting (Birrer, 1988). Signs and symptoms of mild hypovolemic shock may also be exhibited, related to the large volumes of fluid loss and pooling of peripheral blood (Carney, 1989).

Hayes (1984) notes that the complaint of thirst is one of the earliest symptoms of heat exhaustion. Body temperature may reach 105 degrees Fahrenheit or be completely normal (Birrer, 1988; Kunkel, 1986). Vital signs reflect the individual degrees of dehydration and circulatory strain. As total fluid deficit increases, compensatory efforts, such as tachycardia and hypotension may be observed (Barrett, 1991; Knochel, 1989).

If the victim is sodium depleted from excessive free water loss, impaired judgement and disorientation may be observed (Caldroney, 1986; Knochel, 1989; Yarbrough, 1991). Poor cerebral perfusion results from a reduced circulating volume, while cerebral edema, the

consequence of fluid and electrolyte shifts, may complicate this picture. The amount of sweat the patient exhibits will vary, depending on the degree of dehydration (Birrer, 1988). Patients suffering from water depletion may have dry skin. However, when their salt losses are greater, skin will remain clammy (Yarbrough). Serum sodium values will depend on the degree of salt depletion lost via the sweat (Hayes, 1984).

Treatment is symptomatic, guided by the same fundamental, common-sense therapies discussed earlier for the milder forms of heat stress (Carney, 1989). Adequate fluid and electrolyte repletion promotes a generally uncomplicated recovery (Yarbrough, 1991). Assessment of pulse, blood pressure, orthostatic changes, urine output and laboratory values, such as sodium, blood urea nitrogen, and hematocrit assist health care providers in determining volume status requirements. Heart rate and rhythm, as well as oxygen requirements, should be monitored continuously to ensure appropriate interventions and adequacy of therapy (Carney, 1989).

Heat Stroke

Assessment

Diagnosing heatstroke can be problematic. There are no specific tests to confirm the diagnosis. It involves a process of elimination, based on clinical symptoms (Yarbrough, 1991).

Traditionally, heatstroke has been defined to include three common elements: coma, loss of sweat production, and high body temperature. Caldrony (1986) cautions providers not to consider these three criteria absolute when diagnosing possible heatstroke. Eighty percent of those affected have sudden mental status changes (Yarbrough, 1991). Statistics indicate that coma is present in only 70% of cases. Sweating can be observed in over half of exertional heatstroke victims (Caldrony). Even temperatures of 106 degrees Fahrenheit or greater cannot be used alone to diagnose heatstroke. Higher core temperatures can be tolerated by well-trained and acclimated individuals. Furthermore, temperatures below 106 degrees Fahrenheit may not truly reflect the victim's highest temperature, if enough time has lapsed to allow it to fall between the patient's collapse, and the initiation of treatment (Cummings, 1983).

Yarbrough (1991) notes that up to 20% of patients with heatstroke experience a prodrome. This is evidenced by confusion, delirium, lethargy, and combativeness. Prodrome can persist for several days, especially in the elderly population (Cummings, 1983). Symptoms can include giddiness, anorexia, nausea, muscle cramps, extreme thirst, and delirium (Hayes, 1984).

Neurological Changes

The key to recognizing heatstroke lies in recognizing mental status changes that can occur anywhere on the continuum from confused to comatose (Cummings, 1983). It is no surprise that neurological manifestations are predominant signs of this disorder. The brain is especially sensitive to changes in electrolytes and cellular edema (Posey & Caruso, 1986). A full range of neurological signs are attributed to diffuse brain injury, cerebral edema and increased intracranial pressure (Caldronney, 1986).

On admission, the patient may have fixed or dilated pupils from cerebral edema, and this should not be interpreted as a sign of brain death (Anderson et al., 1983). These changes usually reverse as body temperature is returned to normal and fluids shift

(Reed & Anderson, 1986). Mental status changes which persist, despite lowering of core temperature, carry a poor prognosis, indicative of permanent central nervous system damage (Jacobson, 1992; Reed & Anderson).

Electrolyte Changes

Electrolyte abnormalities are observed, especially in sodium and potassium levels, but there is no typical pattern (Cummings, 1983). Values are usually normal on admission, but change over the next few hours, reflecting the severity of cell damage throughout the body (Cummings). Hyperkalemia from rhabdomyolysis, renal failure and/or metabolic acidosis can be life-threatening (Anderson et al., 1983). Interventions to decrease potassium include hemodialysis, and administration of insulin, glucose, calcium, kaexylate enemas and intravenous alkali solutions. Hypokalemia, on the other hand, is more frequently observed in patients with classic heatstroke who are more inclined to be taking non-potassium sparing diuretics. Hypocalcemia may also be evident in those suffering from exertional heatstroke due to a calcium shift into injured skeletal muscle (Anderson et al., 1983).

Renal Changes

Renal failure may develop in heat stroke victims

related to volume depletion and hypotension. The first urine produced by patients with heatstroke is generally small in amount, brown and turbid, described by Yarbrough (1991) as "resembling machine oil" (p. 88). Acute tubular necrosis can result from dehydration, hypotension, "hot kidneys", and an unmet increase in metabolic demand (Cummings, 1983). Kidney function can also be impaired if enough muscle tissue is destroyed (rhabdomyolysis), releasing myoglobin, which clogs renal tubules and impairs filtering capabilities (Cummings). Seizures and shivering associated with cooling measures can also contribute to muscle hyperactivity and rhabdomyolysis (Kunkel, 1986).

Liver Changes

Cellular hypoxia from thermal damage to endothelial cells in the liver causes abnormal function and coagulopathies, evidenced by decreased platelets (Cummings, 1983). Purpura, petechiae, conjunctival hemorrhage, and bleeding in the gastrointestinal tract, lungs, kidneys and other major organs may be observed (Kunkel, 1986). Disseminated intravascular coagulation is rare. However, coagulopathies may result from the release of tissue thromboplastin caused by thermal damage (Caldronney, 1986).

Severe liver damage, evidenced by "marked hepatocellular necrosis", can be a primary cause of death (Caldroney, 1986, p. 48S). Liver function test results tend to peak 48 to 72 hours after the insult. Serum aspartate aminotransferase (AST) values greater than 1000 International Units appear to carry a poor prognosis due to the extensiveness of liver damage (Reed & Anderson, 1986).

Cardiovascular Changes

Caldroney (1986) notes that circulatory findings vary with age, initial volume status, and severity of heat. He adds that younger patients tend to become hyperdynamic (increased heart rate, increased cardiac output, and decreased systemic vascular resistance), while elderly patients are hypodynamic (increased heart rate, decreased cardiac output and decreased systemic vascular resistance). By the time elderly patients are seen, massive volume deficits may exist, requiring hemodynamic monitoring to differentiate pump dysfunction from volume depletion (Caldroney, 1986). According to Jacobson (1992), most heatstroke patients have ST-segment or T wave abnormalities on electrocardiograms (p. 322).

Gastrointestinal Changes

Diarrhea and vomiting are commonly observed in heatstroke patients. Upper gastrointestinal bleeding may occur related to coagulopathies and severe retching or vomiting (Anderson et al., 1983). Pancreatitis has also been observed, attributed to cellular anoxia and severe acidosis (Anderson et al.; Jacobson, 1992).

Pulmonary Changes

Because respiratory drive is stimulated by hyperthermia and the resultant metabolic acidosis, heatstroke victims usually present with a profound hyperventilation (Anderson et al., 1983). Vascular congestion and pulmonary infarction from endothelial injury and cell hypoxia may also contribute to pulmonary damage (Anderson et al.; Jacobson, 1992).

Treatment

The same "golden hour" approach to treating trauma victims can be applied to patients with heatstroke (Iced Peritoneal Lavage, 1990). By reducing body temperature to below 39 degrees Celsius within the first hour, mortality (directly related to the duration of high body temperature) decreases by half (Iced Peritoneal Lavage; Reed & Anderson, 1986). Therefore, the basic goal of treatment in heat-related disorders is to decrease the body temperature as soon as

possible.

In the pre-hospital setting it is best to simply move the patient to a cooler place, undress to allow air to circulate, douse with cool fluids, apply ice packs (if available) to the neck, groin and axillae, and fan the patient (Carney, 1989). In deciding upon the best route of transfer, consideration of evacuation by helicopter offers two advantages: rising to cooler air and wind speeds up to 100 miles per hour, which help in cooling the patient (Cummings, 1983).

Basic principles of supportive care guide the management of patients with heat stroke. The A,B,C's of resuscitation, temperature reduction, isotonic fluid replacement, monitoring (cardiac and central line), accurate urine output measurement, and laboratory tests are all essential (Walker, 1986). Acute changes reverse as body temperature returns to normal; but, changes from anoxia or ischemia may not even be observed for 12 to 24 hours (Posey & Caruso, 1986).

Three non-invasive cooling techniques are used separately or in combination to lower body temperature: ice water immersion, fan-sprayed mist, and ice packing. Ice water baths in conjunction with massage is the most rapid method used to decrease high temperatures

(Anderson et al, 1983). Immersion in ice water reduces the core temperature within 10 to 40 minutes due to the high thermal conductivity of water (Yarbrough, 1991). Though seemingly drastic, Walker (1986) advocates this technique, stating "there is no effective substitute" (p.46). The ice water should be stirred frequently to prevent a layer of warm water from forming next to the skin and reducing conduction (Jacobson, 1992).

The same results can be achieved by spraying water on the patient and fanning, either manually or with electric fans (Cummings, 1983). The United States Marine Corps (USMC) uses misting with warm water (to decrease vasoconstriction) in combination with fans, which they claim works five times faster than immersion techniques (Hayes, 1984). Fans keep air currents moving, maximizing evaporation and cooling.

Anderson et al. (1983) discuss a "body cooling unit...which sprays finely atomized water under pressure at 59 degrees Fahrenheit over a nude body" (p. 132). While admitting this method seemed more physiologic for cooling, the authors, because of their lack of experience with this product, were not prepared to endorse the unit.

Walker (1986) advocates ice packing in conjunction

with fans to increase the rate of surface evaporation. Application of ice packs (ice wrapped in towels) to the neck, axillae, and groin allow heat loss through conduction (Roberts, 1992). Large vessels in these regions lie close to the skin's surface and this facilitates cooling. Direct application of ice is not recommended since it can cause frostbite (Zal, 1984). This method, while slower than immersion in lowering temperatures, is safer for obese patients (Anderson et al., 1983).

There are concerns, however, that warrant discussion regarding these cooling methods. Immersion techniques can be a problem when dealing with comatose patients who require airway control and aspiration prevention (Cummings, 1983). This method has other disadvantages. Increased vasoconstriction and shivering can lead to increases in muscle metabolism and temperature. It also can be very difficult to maintain control over a patient in a tub, as well as being extremely uncomfortable for caregivers to work in ice water (Cummings, 1983).

Cummings (1983) claims the same effect obtained with ice water immersion can be achieved from simply poring ice water on the patient, massaging, and

fanning. This minimizes discomfort and addresses safety issues. Massaging is beneficial with any technique because it reduces vasoconstriction, eliminates heat, and promotes the return of cool peripheral blood to internal organs. Cold IV fluids and cooling blankets do not offer good control and should not be used (Yarbrough, 1991).

There is considerable debate reported in the literature regarding the use of wet towels or sheets to cover the patient. Caldrony (1986), Posey and Caruso (1986), and Walker (1986) advocate this method in conjunction with fans to increase the rate of surface evaporation. Kunkel (1986), on the other hand, argues that sheets or towels soaked in water are not an efficient method of lowering temperatures because they impede heat loss. Ultimately, personal experience, training, and the policies/protocols of individual institutions will prevail over which method is employed.

Invasive cooling techniques--gastric, peritoneal, and rectal lavage with iced saline--offer another rapid approach to cooling heatstroke victims. Gastric lavage with iced saline has been successful in reducing core temperatures quickly (Hayes, 1984). Insufficient

control in monitoring the patient's temperature may inadvertently lead to hypothermia when using this technique (Birrer 1988; Yarbrough, 1991). Extreme care must be exercised if the decision is made to perform this intervention.

Research conducted on lab animals suggest peritoneal lavage is the quickest way to cool vital organs, such as the kidney and liver (Iced Peritoneal Lavage, 1990). Peritoneal lavage, a routine procedure in the emergency room, offers rapid cooling and perhaps the best chance for avoiding complications associated with hyperthermia (Iced Peritoneal Lavage). Because of the invasiveness required, it is recommended that this procedure be reserved for comatose patients whose temperature remains elevated at greater than 42 degrees Celsius after 20 minutes of conventional methods, without an appreciable improvement (Iced Peritoneal Lavage).

Some literature suggests the use of iced rectal enemas in lowering temperatures (Birrer, 1988; Hayes, 1984; Kunkel, 1986). Others advise against this because of the potential for water intoxication (Anderson et al., 1983; Posey & Caruso, 1986; Yarbrough, 1991). Hayes (1984) notes that iced saline

enemas interfere with continuous monitoring of rectal temperatures, necessary in preventing hypothermia. Since this treatment modality offers questionable results in lowering body temperature and has obvious disadvantages, a more appropriate method should be pursued.

Alcohol used in ice water or applied directly on the skin can poison children and should be avoided in treating this patient population (Jacobson, 1992; Yarbrough, 1991). Jacobson (1992) relates an incident where an explosion occurred on top of a patient receiving an alcohol rub. The friction vaporized the alcohol, reached a flash point, and exploded. If alcohol must be used, a 1:1 dilution with water is recommended.

Supportive Care

Because there is a three to four degree Fahrenheit difference between body core and peripheral temperature during cooling, core temperature measurements are necessary to determine accurate assessments of heat stress and true body temperature. This can be accomplished by placing a thermistor at least 20 centimeters into a body cavity. The esophagus or rectum may be used (Caldroney, 1986). Roberts (1992)

relies on rectal temperature readings. They have proven more accurate than oral, axillary or tympanic measurements. He suggests this is due to the closeness of rectal circulation to internal organs. Swan-Ganz catheters, if used, have thermistors which also provide core temperature measurement in the heart.

Cooling is generally discontinued when the core temperature reaches 102 degrees Fahrenheit. The temperature is then allowed to normalize on its own (Hayes, 1984). Core temperature measurements should be done every five minutes to prevent overshoot and potential hypothermia (Yarbrough, 1991).

Kunkel (1986) recommends supplemental administration of oxygen because of hypoxia and the hypermetabolic state. He also advocates early endotracheal intubation to ensure protection of the patient's airway.

Classic heat stroke requires conservative fluid replacement, unlike exertional heatstroke, where more intensive fluid needs may be observed (Reed & Anderson, 1986). Though blood pressure may be low on admission, intravenous fluids should be given judiciously to avoid problems that occur as the patient cools down (Cummings, 1983). In classic heatstroke, the drop in

blood pressure is due to dilated superficial vessels; and cooling usually restores the pressure as fluid shifts back into circulation (Reed & Anderson). If large amounts of fluid have been given, there is a risk of overloading the system when fluid in superficial vessels shifts back into the intravascular spaces. For the patient with an unhealthy heart and circulatory system, this can prove disastrous.

Hypotension usually resolves with cooling, rather than fluids, especially in the elderly with nonexertional heat stroke (Yarbrough, 1991). Average total fluid requirement is 1200 milliliters over four hours (Yarbrough). Monitoring via central line or Swan-Ganz can be helpful in preventing fluid overload (Kunkel, 1986). Normal saline (NS), lactated ringers (LR), or D5.45NS may be given depending upon the policy of individual institutions (Kunkel, 1986; Carney, 1989).

Valium is frequently used to control shivering (Cummings, 1983). Walker (1986) notes that morphine sulfate can also be effective in treating shivers as well as increasing vasodilation, which increases heat loss. Chlorpromazine is also effective, but may cause hypotension and must be used cautiously (Anderson et

al., 1983).

Convulsions, common during cooling, increase heat production and interfere with cooling (Reed & Anderson, 1986; Yarbrough, 1991). Valium and dilantin are given most frequently to control seizure activity (Kunkel, 1986), although in severe cases, general anesthesia and pavulon have been used to abolish muscle hyperactivity associated with seizures. Benzodiazepines and phenobarbital to sedate or control seizure activity are contraindicated in patients with potential or actual liver damage (Yarbrough, 1991).

Dantrolene, effective in the treatment of malignant hyperthermia, has been studied for its efficacy in treating heatstroke but found ineffective (Bouchama, Cafegé, Devol, Labdi, El-Assid & Seraj, 1991; Caldroney, 1986; Knochel, 1989). Dantrolene "acts directly on skeletal muscle... thought to increase calcium intake, or inhibit its release...and reverse muscle rigidity and heat production" (Bouchama et al., 1991).

Orser (1992) critiques the methods of the recent studies that indicate a lack of benefit in treating heatstroke patients with Dantrolene. First, were the trial doses adequate? She notes "cellular mechanisms

responsible for the increase in temperature during heatstroke and malignant hyperthermia are different and hence, the amount of dantrolene required to treat these two disorders may not be similar" (p. 1193). Second, she observed that each vial of dantrolene also contains mannitol (three grams) and questioned the role mannitol plays in the overall effectiveness. Her recommendations for future research with dantrolene include appropriate controls for mannitol and using larger doses of dantrolene.

Another concern with the study done by Bouchama and colleagues is that all participants had classic heatstroke. The exclusion of patients with exertional heatstroke, for whatever reason, restricts the conclusions of this study.

Mannitol may prove helpful in encouraging renal blood flow and prevent damage from myoglobinuria, as well as reduce cerebral edema (Anderson et al., 1983; Yarbrough, 1991). If adequate fluids have been given, furosemide may also be used to promote renal blood flow (Hayes, 1984).

An alkaline diuresis is indicated in the treatment of myoglobinuria from rhabdomyolysis. Alkalinization of urine is thought to decrease the nephrotoxicity of

myoglobin (Toto, 1990). This can be accomplished by administering two ampules of sodium bicarbonate per liter of D5/W and maintaining a urine output greater than 50 cc/hr. Adequate fluid resuscitation is necessary to prevent acute tubular necrosis, caused by precipitation of myoglobin in renal tubules (Walker, 1986) and/or shock (Hayes, 1984).

Normalization of temperature is usually accompanied by resolution of neurological symptoms, although patients may remain confused and sleepy (Hayes, 1984). There are minor disagreements regarding the length of time necessary for temperature regulation to stabilize. Hayes (1984) indicates it may take up to seven days for the temperature to stabilize; while others report it can take up to several weeks (Birrer, 1988; Carney, 1989).

Summary

Heat stress represents a broad spectrum of heat-related disorders, whose effects are cumulative and progressive. Symptoms can range from mild edema to life-threatening organ failure. Prompt recognition and rapid intervention can mean the difference between life and death.

CHAPTER FOUR

Prevention

There is almost universal agreement in the literature that prevention is the best cure for heat stress. Caldrony (1986) cites the old adage "an ounce of prevention is worth a pound of cure." This is especially true in victims of heat stress.

Prevention through education and awareness should be an important part of orientation programs at work, school and/or recreation facilities. Programs to emphasize important aspects of preventing heat stress can be developed to target workers, children, senior citizens, and other high risk groups.

Personalization, having individuals speak from personal experience, is an effective means of communicating the importance of prevention to others (Barrett, 1991). This technique is useful because it carries more weight for those inclined to be skeptical (Barrett).

The best defense against heat stress is staying well-hydrated (Cummings, 1983). Drinking plenty of fluids and avoiding alcohol are good practices to follow (Hayes, 1984). Replacement fluids for victims of heat stress should be cold if available, because

they are absorbed more quickly and cool internal structures (Zal, 1984). Cummings (1983) offers excellent information on maintaining adequate hydration. Though originally addressed to hikers and athletes, his advice is universally applicable. Urine should always be clear. If it is not, you are not drinking enough.

Thirst is a poor indicator of hydration needs. By the time an individual senses thirst, up to a 50% volume deficit may exist (Barrett, 1991). Hydration requirements must be assessed prior to heat exposure and continue throughout, with an emphasis on maintaining rather than making up a deficit. According to Jacobson (1992), the rule of thumb for adequate intake is one and a half times the amount that quenches thirst.

Information must be disseminated, not just to workers and athletes, but to the general public that thirst is not a reliable indication of dehydration. By the time this has occurred, there can be up to a 50% deficit in body fluids (Barrett, 1991). Those in supervisory positions must be informed of the potential hazards and warning signs (Cummings, 1983). Screening individuals to identify those at greater risk is also

important, as well as assuming individual responsibility for decisions and actions (Barrett, 1991).

The "most common cause" of hyperthermia is exercise (Vander, 1990). Surveillance teams at races, marathons, sporting events, or any outdoor event where heat stress may be a factor should be well-versed on the potential for problems and be prepared with adequately trained personnel to render prompt diagnosis and intervention (Walker, 1986). Weather should be monitored to avoid peak heat in timing or scheduling events. Physical activity at work or play should be avoided during hours where temperature and humidity are likely to be highest (Walker, 1986; Zal, 1984).

When heat exposure is anticipated, especially long term, the process of acclimatization can be crucial. Caldwell (1991) attributes the success of keeping heat-related casualties during Operation Desert Shield and Storm at a minimum to the military's effort to acclimatize soldiers both before and after deployment, and the fact that full duty schedules were not essential immediately upon arrival in country.

California's Mojave Desert serves as a regular training ground for desert warfare (Dreyfuss, 1992).

Acclimatization began here prior to deployment during the Persian Gulf War. Marines spent several days in large warehouses in the California desert to begin adjusting to high temperatures, where they were encouraged to participate in light exercise or sports activities, since this improves the body's ability to sweat (Dreyfuss, 1991).

Americans drew on Israel's experiences in the Six Day War of 1967 and the Yom Kippur War of 1973 in planning for heat prevention (Dreyfuss, 1992). Drinking was made mandatory. Briefing teams were sent to deploying units to instruct on recognition of early signs and symptoms and the overall prevention of heat-related injuries (Dreyfuss).

Caldwell (1991) reports the importance of training in dealing with the natural threats of heat stress and dehydration and chemical defense clothing during the Persian Gulf War. Few problems were actually encountered due to the importance placed on frequent reminders to drink plenty of water, stay cool, and avoid work and play during the hottest time of the day. Statistics gathered during this military engagement, however, only reflect patients seen in a treatment facility, and are not indicative of milder forms of

heat stress which may have been treated in the field (Dreyfuss, 1991).

Clothing is also an important consideration in countering the effects of heat. Light-colored, lightweight loose clothing averts heat gain and allows better ventilation for cooling (Zal, 1984). Hats provide protection from heat and ventilation to keep heads cool (Zal).

The Centers for Disease Control (1981) recommends that architects and builders consider criteria for design and construction that would "maximize air movement and provide improved insulation and air handling equipment" (p.278), especially in urban areas. Hayes (1984) advocates using the good neighbor policy: check frequently on the elderly and chronically ill during seasonal heat waves and restrict children to playing inside, limiting their exposure to the stresses of heat. Communities have established public service programs to offer fans and air conditioning to high risk groups, such as senior citizens (Parks & Calabro, 1990).

Summary

Prevention plays a critical role in dealing with the effects of heat stress. Much can be done to

control or eliminate unfortunate complications associated with heat-related disorders. With advanced planning and common sense, heat stress can generally be avoided (Jacobson, 1992).

Summary

Providing care for the patient suffering the effects of extreme heat is a challenge for health care providers. The effects of heat stress are progressive and potentially life-threatening. Trained athletes and acclimated workers are affected as often as "ordinary" people.

An understanding of the basic physiology of thermoregulation, acclimatization, and the mechanisms of heat dissipation plays an important role in promoting early recognition and treatment. An accurate assessment and rapid intervention is essential to avoid adverse effects and possible death.

Prevention is also critical factor when discussing heat stress. Knowledge can have a positive effect, since heat stress can usually be prevented. Understanding prevention techniques and risk factors gives control to each individual over this potentially devastating illness.

CHAPTER FIVE

The Role of the Clinical Nurse Specialist

The critical care nurse specialist is defined by the American Association of Critical Care Nurses (AACN) to be an expert in critical care nursing, as a result of study and supervised practice at the masters or doctoral level (AACN Position Statement, 1987). Delineated in this position statement are five major functions of the Clinical Nurse Specialist (CNS) role: advanced practitioner, educator, consultant, researcher, and manager.

Advanced Practitioner

The advanced practitioner role was "established to improve the quality of patient care and nursing practice" (Koetters, 1989, p. 107). Providing direct patient care places the CNS in an ideal position, working directly with patients, to identify practice issues and problems. Quality of care issues receive firsthand attention.

Maintaining a clinical practice lets the staff nurses know that this is an important subrole, one not willing to be lost because of administrative pressures. It also establishes a foundation for building trust, confidence and a camaraderie with staff nurses. The

CNS at the bedside providing care, reassures staff members that the CNS does indeed possess expert knowledge, skills, and resources that are available to them. In the direct care capacity, the CNS becomes a role model.

It is not often that victims of heat stress are admitted to critical care units. They are usually evaluated and treated in the emergency room and released. However, in extreme cases of heat stroke, patients do find their way into intensive care units. Infrequent heat-related admissions place staff nurses at a disadvantage in providing care. Patients admitted with heat-related disorders can present a vast array of complications.

As an expert practitioner, the CNS possesses the necessary skills and knowledge to provide excellent patient care for these individuals. The CNS is comfortable with the complex pathophysiological manifestations of heat stroke, and can be instrumental in planning care as a guide for other staff members, as well as developing protocols related to heat stress victims.

Educator

The role of educator, along with being an expert

practitioner, is one of the more traditional expectations of the CNS. Education is perhaps the strongest area for the CNS to practice in relation to patients suffering from heat stress. Staff, patient, and student educator are three components of this position described by Priest (1989).

Ideally the CNS will evaluate staff educational needs regarding heat stress during orientation and through conducting needs assessments. Recognized as having expert knowledge in this area, the CNS should participate in planning and providing inservice programs, continuing education offerings, and safety briefings based on these evaluations and assessments.

Because heat stress victims are primarily treated in the emergency room, the CNS must set the tone for a collaborative practice to share information throughout the facility. Developing, presenting and sharing educational programs with all disciplines would promote this practice.

When providing direct patient care, the educational focus for the CNS is on the patient and family. Prevention is the best weapon against heat-related disorders and the CNS is in an excellent position to initiate discharge teaching and planning.

It must include not only prevention techniques, but signs, symptoms, and early recognition of heat stress as well.

Family and significant others are important to include in this educational process. Once a victim of heat stress, the individual is at increased risk for heat-related injury in the future and may need to rely on the recognition and assessment skills of these individuals, since their judgement may become impaired at very early stages.

Consultation

Consultation is an important subrole of advanced nursing practice offering "education, clarification, diagnostic formulation, and additional problem-solving strategies" (Barron, 1989, p. 126). It is a role that depends on expertise and a thorough understanding of the process (Edlund, Hodges, & Poteet, 1987).

Providing consultative services on the subject of heat stress will be an important role for the advanced practitioner. Ideally, the consultant functions to mobilize internal and external resources to deal with problems and change efforts (Edlund, et al., 1987).

As a military service member, providing true external consultation is not an expectation. It cannot

be discounted, however, due to the liaison efforts of health care providers between military bases and local communities. Participation in disaster drill and/or shared educational offerings are examples.

Heat prevention is an integral part of safety and mobility training. The CNS with knowledge in this area could actively market her expertise. External consultation could be construed to encompass the base installation or command. An example of this would be consulting with mobility instructors on teaching prevention of heat related illness for military members.

On the other hand, internal consultation, not only for individual units, but the entire facility is an expectation. Non-health care providers often are responsible for organizing training and education programs and must rely on experts in related fields for professional advice and information.

As noted earlier, admission of heat stress victims may not be routine enough to instill a level of confidence on the part of nursing staff. The CNS would be able to provide expertise and guidance in providing quality care to these individuals. Working with the consultee, protocols and care plans can be developed,

not only for the present, but future reference as well. It is important for the CNS acting as a consultant to make the consultee aware that services are always available.

Researcher

Though mandated by the American Nurses Association (ANA), the role of research still lacks support or direction for most nursing practices (McGuire & Harwood, 1989). McGuire and Harwood (1989) identified three separate levels of research activities for the CNS. Level one establishes minimum criteria for practice; level two pertains to post-master's experience, while level three describes a more advanced practice. These levels provide a guide to the CNS in assessing individual standing and expectations.

The CNS is in an ideal position to identify nursing practice problems in the clinical setting related to the management of heat-related disorders. Entry level nurse researchers will also be able to devise research questions based on these problems.

The second level of research activity described by McGuire and Harwood (1989) involves participation in research without primary responsibility.

Collaborating with physicians, other practitioners or clinical staff in research projects to replicate studies, or being involved with current projects, are also appropriate research activities at this level. Gathering data for research projects, evaluating methods of practice, and monitoring patient outcomes are activities in which the CNS would ideally be involved. Environmental services attached to individual facilities can be helpful in providing data for possible studies.

There are many research questions that could be explored by the CNS. Would larger doses of dantrolene prove effective in quickly reducing high temperatures associated with heatstroke? Which cooling method achieves the quickest lowering of temperature: ice water immersion versus spray mist techniques with fans versus ice packing with fans? How many victims of heatstroke suffer long-term residual effects? How many health care providers are willing to perform iced peritoneal lavage? What is the impact of the CNS in the care and outcomes of patients with heat-related disorders?

Maintaining a level of currency with available literature is an important function for the advanced

practitioner. Being able to convey that information to other health care providers and staff is fundamental for the CNS.

Manager

The CNS role falls victim to close scrutiny due to traditionally being viewed as non-revenue producing and expensive for a non-direct care position (Peglow, Klatt-Ellis, Stelton, Cutillo-Schmitter, Howard & Wolff, 1992). Administrations may respond by eliminating these positions or moving the CNS to line positions.

The CNS, however, can compete in this changing environment. Gournic (1989) emphasizes that clinical leadership is a major component of the CNS role, which makes them a natural choice for management positions. She adds that careful structuring of line positions can make it possible for the CNS to maintain both practice and clinical leadership roles. Role success depends on clear definitions and interpretation, as well as expectations (Arford & Olson, 1988).

Managing and influencing patient care activities, as well as acting as a change agent to create an environment of empowerment are important aspects of the CNS role. This CNS will develop the role of manager by

requesting and assuming broad responsibilities. Committee memberships and involvement is an expectation in the military. These are looked upon as growth opportunities which challenge the individual to develop leadership and management skills.

Establishing a collaborative relationship with the nurse manager can open the door to a more active role in the administrative functioning of the unit. Assuming responsibility for skills verification, budget and scheduling preparation, and written policies and procedures involving quality of patient care (standards of care and standards of practice) are ways this CNS will broaden the managerial role. This participation will hopefully create an atmosphere of ownership, understanding, and responsibility, which can only strengthen the integrity of the unit and benefit everyone concerned.

Summary

There are many dimensions to the role the CNS can play in caring for patients with heat stress. Adaptability combined with expert knowledge enables the CNS to be a positive force in influencing outcomes related to heat stress victims in the trauma/critical care setting.

References

- American Association of Critical Care Nurses (1987).
The Critical Care Clinical Nurse Specialist: Role Definition. Aliso Viejo, California: author.
- Anderson, R. J., Reed, G., & Knochel, J. (1983).
Heatstroke. Advances in Internal Medicine, 28, 115-140.
- Arford, P. H. & Olson, M. (1988). A structural perspective of nurse manager and clinical nurse specialist collaboration. Clinical Nurse Specialist, 2(3), 119-126.
- Barrett, M. V. (1991). Heat stress disorders: old problems, new implications. American Association of Occupational Health Nursing, 39(8), 369-380.
- Barron, A. (1989). The cns as consultant. In A. B. Hamric & J. A. Spross (Eds.), The clinical nurse specialist in theory and practice (pp. 125-146). Philadelphia: W. B. Saunders Company.
- Birrer, R. B. (1988). Heat stroke: don't wait for the classic signs. Emergency Medicine, 20(12), 8-10, 12-3, 16.
- Bouchama, A., Cafege, A., Devol, E. B., Labdi, O., El-Assil, K., & Seraj, M. (1991). Ineffectiveness of dantrolene sodium in the treatment of heatstroke.

- Critical Care Medicine, 19(2), 176-80.
- Caldronney, R. D. (1986). Heat-induced illness.
Hospital Practice, 21(6), 48M-N, 48R-T.
- Caldwell, J. A. Jr. (1992). A brief survey of chemical defense, crew rest, and heat stress/physical training issues related to operation desert storm.
Military Medicine, 157(6), 275-82.
- Carney, M. (1989). When the heat is on. Emergency, 21(6), 24-5, 29.
- Centers for Disease Control (1981). Heatstroke---United States, 1980. Morbidity and Mortality Weekly Report, 30(23), 277-279.
- Centers for Disease Control (1984). Heat-Associated Mortality---New York City. Morbidity and Mortality Weekly Report, 33(29), 430-432.
- Cummings, P. (1983). Felled by the heat...heatstroke. Emergency Medicine, 15(12), 94-8, 103-4, 109-10.
- Dreyfuss, I. (1991). Desert shield: military wins battle against heat injury. Physician and Sportsmedicine, 19(6), 141-5.
- Edlund, B. J., Hodges, L. C., & Poteet, G. W. (1987). Consultation: doing it and doing it well. Clinical Nurse Specialist, 1(2), 86-90.
- Gournic, J. L. (1989). Clinical leadership,

- management, and the cns. In A. B. Hamric & J. A. Spross (Eds.), The clinical nurse specialist in theory and practice (pp. 227-248). Philadelphia: W. B. Saunders Company.
- Guyton, A. C. (1991). Textbook of medical physiology (8 ed.). Philadelphia: W. B. Saunders.
- Hayes, K. B. (1984). Dealing with heat injuries. RN, 47(7), 41-4.
- Iced Peritoneal Lavage for Heat Stroke (1990, May 30). Emergency Medicine, pp. 83-84.
- Jacobson, S. (1992). The ill effects of heat. Emergency Medicine, 24(7), 313-4, 317-8, 321-4.
- Knochel, J. P. (1989). Update on summer heat syndromes. Patient Care, 23(11), 87-90, 95, 98.
- Koetters, T. (1989). Clinical practice and direct patient care. In A. B. Hamric & J. A. Spross (Eds.), The clinical nurse specialist in theory and practice (pp. 107-123).
- Kunkel, D. B. (1986). The ill effects of heat: environmental causes part 1. Emergency Medicine, 18(14), 173-4, 177, 181-2.
- McGuire, D. B. & Harwood, K. V. (1989). The CNS as researcher. In A. B. Hamric & J. A. Spross (Eds.), The clinical nurse specialist in theory and practice

- (pp. 169-203). Philadelphia: W. B. Saunders Company.
- Orser, B. (1992). Dantrolene sodium and heatstroke [Letter to the editor]. Critical Care Medicine, 20(8), 1192-1193.
- Parks, F. B. & Calabro, J. J. (1990). Hyperthermia: performing when the heat is on. Journal of Emergency Medical Services, 15(8), 24-25, 27-32.
- Peglow, D. M., Klatt-Ellis, T., Stelton, S., Cutillo-Schmitter, T., Howard, J., & Wolff, P. (1992). Evaluation of the clinical nurse specialist practice. Clinical Nurse Specialist, 6(1), 28-35.
- Posey, V. M. & Caruso, C.C. (1986). Life-threatening heat-related emergencies. Dimensions of Critical Care Nursing, 5(4), 216-27.
- Price, B. (1984). Mad dogs and englishmen...heat exhaustion. Nursing Times, 80(32), 16-9.
- Priest, A. R. (1989). The cns as educator. In A. B. Hamric & J. A. Spross (Eds.), The clinical nurse specialist in theory and practice (pp. 147-167). Philadelphia: W. B. Saunders Company.
- Reed, G. & Anderson, R. J. (1986). Emergency: heatstroke. Hospital Medicine, 22(8), 19-21, 25, 29-30.

- Roberts, W. O. (1992). Managing heatstroke: on-site cooling. Physician and Sportsmedicine, 20(5), 17-8, 20, 28.
- Toto, K. H. (1990). Rhabdomyolysis. In B. C. Mims (Ed.), Case studies in critical care nursing (pp. 328-339). Baltimore, MD: Williams & Wilkins.
- Vander, A. J., Sherman, J. H., & Luciano, D. S. (1990). Human physiology: the mechanism of body function (5th ed.). New York: McGraw-Hill.
- Walker, M. (1986). When the going gets hot. Nursing Times, 82(32), 44-7.
- Yarbrough, B. E. (1991). Heat-related disorders. Hospital Medicine, 27(6), 81-3, 87-91, 117-20.
- Zal, H. A. (1984). Recommended program for employees exposed to extremes of heat. Occupational Health Nursing, 32(6), 293-6.