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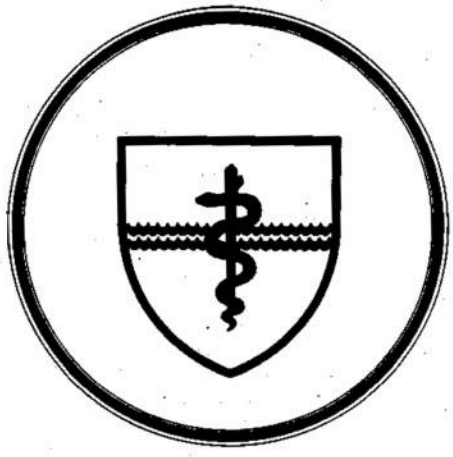

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HYPOTHERMIA

AN EDUCATIONAL MANUAL FOR INSTRUCTION OF THE FLEET DUTY CORPSMAN ACCOMPANYING PERSONNEL PERFORMING OPERATIONS IN COLD WATER OR COLD WEATHER

by

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SUMMARY PAGE

PROBLEM:

To educate the diving corpsman and others who will have medical duties relating to operations performed in cold weather and cold water.

FINDINGS:

Presentation is made of the normal thermoregulatory mechanisms, the pathophysiology of hypothermia, the clinical presentation, treatment and prevention. A summary abstract is provided for easy review. A full topic bibliography is provided for the readers' reference.

APPLICATION:

The material presented will be of value in preventing morbidity from hypothermia casualties in cold water diving operations.

ADMINISTRATIVE INFORMATION

This report was submitted for review in October 1980 and approved for publication in November 1980. It is designated as NavSubMedRschLab Report No. 943.

ABSTRACT

Hypothermia connotes the abnormal lowering of body core temperature (the internal temperature of the body; that of the core organs) the effects of which are in a gradient from only a mild decrease in motor and cognitive functions to the severe reactions of cardiac and respiratory failure. Hypothermia becomes important to the corpsman who will accompany personnel into the thermally stressful environments of cold weather and cold water.

This presentation is designed to provide the corpsman with a basic understanding of the body's normal thermoregulatory mechanisms, their methods of adaptation to cold stress and the clinical syndrome which results. Knowledge of the basic pathophysiology will allow the corpsman to interpret the subtle objective indicators of hypothermia and to recognize the vague subjective indicators as voiced by the victim. This background will facilitate prompt treatment and relief of a potentially dangerous malady.

PREFACE

The purpose of this educational manual is to provide an information and reference source for training fleet duty corpsmen in preparation for their deployment with personnel performing operations in cold water or cold weather. This includes the accidental immersion of a man overboard as well as the carefully planned diving operations normally performed in the fleet.

There are two sections contained herein. Each has a specific purpose and each is meant to stand alone. The first section is intended to provide the instructor with a wide base of information regarding all aspects of hypothermia. This will allow the instructor to pass this knowledge on to the corpsman student in a comprehensive fashion utilizing the second section as a reference handout with which the student may follow along with the lecture and also retrieve for easy future reference. The depth with which the topic is presented in section one is intended to allow the instructor to be as fully versed in the subject as possible so that both instruction and question answering will be facilitated.

A comprehensive bibliography is appended for the convenience of the reader's future reference.

D.C.A.

TABLE OF CONTENTS

SECTION ONE: INSTRUCTOR FAMILIARIZATION 1

CHAPTER I INTRODUCTION 3
CHAPTER II NORMAL THERMOREGULATORY MECHANISMS 7
CHAPTER III PATHOPHYSIOLOGY OF HYPOTHERMIA 15
CHAPTER IV CLINICAL PRESENTATION 30
CHAPTER V TREATMENT 37
CHAPTER VI PREVENTION 48

SECTION TWO: CORPSMAN REFERENCE HANDOUT 53

BIBLIOGRAPHY 66

SECTION ONE

INSTRUCTOR FAMILIARIZATION

CHAPTER I

INTRODUCTION

Hypothermia is defined as an abnormal lowering of body core temperature (the internal temperature of the body; that of the vital organs), the effects of which are in a gradient from only a mild decrease in motor and cognitive functions to the severe reactions of cardiac and respiratory failure. The purpose of this presentation is to serve as an educational guide for Navy Corpsmen who would have occasion to encounter hypothermia as they accompany personnel who will be subject to immersion in cold water and exposed to cold weather. Diving operations obviously present high risk. The diver and also the ancillary personnel must be considered. This includes the tenders, supervisors and stand-by divers. As reported by the 1980 International Hypothermia Conference and Workshop at the University of Rhode Island, the past eight years have seen 917 SCUBA fatalities, one third in water of less than 65°F and, of those, one quarter were probably victims of hypothermia.

'Can't people tell when they're getting cold?' one might ask. Yes; but when one is diving in cold water, one expects to feel cold, yet this is not a quantitative measure of body heat loss and the effects of hypothermia are insidious, not readily detectible by the casual observer. The early effects are sometimes

subtle. In addition to early cognitive functional decrements, there is a dramatic decrease in motor function due to the compensatory mechanism of vasoconstriction seen in hypothermia. Hypothermia also increases susceptibility to decompression sickness, carbon dioxide toxicity (partially due to the decreased effectiveness of the carbon dioxide scrubbers in cold water) and oxygen toxicity.

Heat is extracted from the body by the colder environment. If metabolic heat production can keep pace with external losses, there is homeostasis. If it cannot, then shivering and vasoconstriction are the next responses but are only stop-gap measures as body energy is quickly drained with continued exposure. As will be discussed, body heat is gained through production of metabolic heat as well as shivering, is lost through conduction, evaporation, convection and radiation and is conserved by vasoconstriction and protective clothing.

Interestingly, water at 80°F causes the same heat loss as 42°F air. 92°F is the magic temperature for water; the temperature at which one can remain in homeostasis - heat production can keep pace with environmental heat loss. Below 92°F, the body will eventually begin to lose more heat than it can produce. Exercise will increase heat production but, unfortunately, will also increase heat loss and at a greater rate. One can maintain body heat in 60°F water with maximum shivering but for only a short period of time until energy stores are depleted.

Consider, for a moment, a common fleet scenario of a diver performing cold water diving operations: he surfaces after breathing

cold gases in cold water and working hard on the bottom - he's cold. During his surface interval, he takes a hot shower and drinks some hot coffee. He feels better now and is ready for another dive. On the second dive, his performance is less than earlier that same day; he has trouble operating simple tools, forgets items on a checklist, his speech becomes garbled and he is brought to the surface by his tenders with thoughts of carbon dioxide build-up or other common diving malady. There is no thought of hypothermia. Let us see what might have really happened. The diver became chilled on the first dive, invoking vasoconstriction of his extremities as the primary defense mechanism against hypothermia, thereby allowing his extremities to chill while preserving the core temperature. While on the surface, however, the hot shower acts to break the vasoconstriction and produce vasodilation. This has two effects; first to allow warm blood in the core to be cooled by the relatively cold extremities and, secondly, the diver will be vasodilated when reentering the water, thereby becoming colder quicker. The result is a hypothermic diver manifesting decreased mental and physical functioning without realizing it himself.

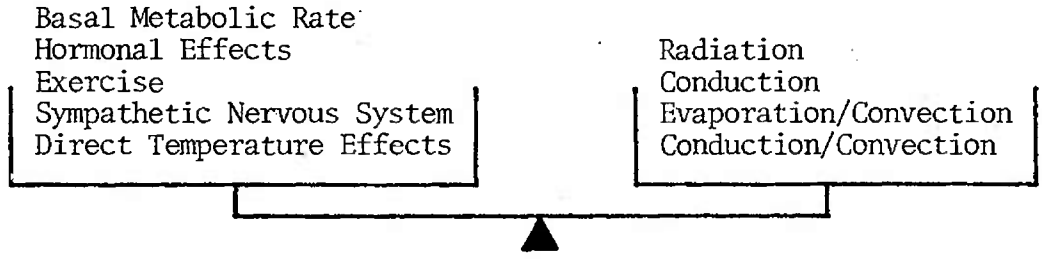
The effects of hypothermia vary with the individual, depending, in part, on body build, body fat content, state of nutrition, physical conditioning and anaerobic capacity. The corpsman should have an understanding of the body's normal thermoregulatory mechanisms of heat production and conservation, the subtle signs and symptoms of hypothermia, treatment modalities

and methods of prevention. Each of these topics will be discussed in detail.

CHAPTER II

NORMAL THERMOREGULATORY MECHANISMS

The temperature of the body remains nearly constant with only 1°F diurnal variation. Heat is continually being produced as a by-product of normal metabolism and is being constantly lost to the surroundings. When these two processes occur at the same rate, the body is said to be in thermal equilibrium. Factors which affect heat production are the basal metabolic rate, the effects of hormones, exercise, Sympathetic Nervous System effects and the direct effect of temperature on cellular metabolism. Factors involved in heat loss are radiation, conduction, evaporation/convection and conduction/convection. We shall examine each of these factors individually as they affect normal temperature homeostasis as well as their central regulation.



A. Heat Production

Metabolism is simply the conversion of the raw materials contained in food to useful materials and energy, producing heat as a by-product. The metabolic rate determines the amount of heat produced. Only about 25% of the energy stored in food is

eventually available for use by the body, the remaining 75% becomes heat. The Basal Metabolic Rate (BMR) is the lowest metabolic rate of a given individual at rest and is specific for each person. The metabolic rate can, however, be increased by a number of factors.

Exercise causes the most dramatic effect on metabolic rate. Short bursts of maximal muscle contractions can liberate as much as a hundred times the normal amount of heat. Overall, maximal muscle exercise can increase the rate of heat production to fifty times normal for only a few seconds or twenty times normal for several minutes. Jumping ahead, one can readily see that shivering, or maximum muscle activity, can rapidly deplete the body of available energy stores. If shivering were the only heat producing mechanism standing between normal thermal homeostasis and hypothermia, then when exhaustion ensued, the individual's heat loss would not be compensated for and severe hypothermia would result.

Age is also a factor in Basal Metabolic Rate, but mentioned here only for general information since the BMR is relatively constant between the ages of 18 and 50. The BMR is highest for the very young and lowest for the aged.

The thyroid gland is the metabolic carburetor for the body. When it maximally secrets the hormone, thyroxine, the metabolic rate can be increased up to one hundred times the basal rate. In contrast, loss of thyroid function would decrease the metabolic rate by only one-half. This illustrates the basic function of

the thyroid gland; to increase the metabolic rate in response to body needs. (This makes sense since the Basal Metabolic Rate is supposed to be the lowest metabolic rate allowing normal functioning at rest.) Thyroxine is one of the hormones released in response to cold as well as other stresses. There is, however, a lag time between stimulus and secretion and between secretion and the eventual effect of thyroxine on the metabolic rate.

Sympathetic Nervous System stimulation occurs as a result of exposure to cold. This causes the release of other metabolically active hormones such as epinephrine, norepinephrine and growth hormone which can collectively increase the metabolic rate as much as 100%. Although this effect seems small in comparison with the others above, the Sympathetic Nervous System also controls vascular tone which, as will be explained later, is the most important mechanism for preserving whatever heat is produced in the cold environment.

Anecdotally, the male sex hormone, testosterone, can increase the metabolic rate by 10-20% but this effect is insignificant when taken in light of the other more powerful metabolic controls.

Direct local effect on cells is a major factor for only those cells in closest proximity to the surface; that is, those cells maximally affected by environmental cold. These cells are the skin, subcutaneous tissue, fat (which already has a low metabolic rate) and structures contained in these superficial layers. These structures include the nerve endings which receive pain and temperature stimulation. If their metabolic rate is

severely lowered, numbness will result from non-functioning of these receptors. One should remember that the lungs are also in direct contact with the environment by virtue of breathing the cold air or gases. This becomes extremely important when one realizes that vasoconstriction to preserve body heat cannot occur in the lungs since the entire cardiac output must pass by these cold air channels. The lungs are a major source of heat loss and can be a major avenue for rewarming therapy as we shall see later.

B. Heat Loss

The methods of heat loss are radiation, conduction, evaporation/convection and conduction/convection. Heat loss by radiation is in the form of infrared heat rays (the same infrared rays detected by night vision devices used in sniping and other night personnel-monitoring operations) given off into the surrounding air. A nude man will lose about 60% of his total heat loss by radiation. Every object in his surroundings is also radiating infrared rays proportional to its temperature. Thus, if the temperature of the surrounding objects were greater than the man's, such as in a boiler room or in the hot summer sun, the net effect would be a greater infrared radiation by the surroundings than by the man resulting in a net heat *gain* by the man. So, it works both ways. In diving operations, however, the surrounding chamber atmosphere is rarely hotter than the 98.6°F of man. Heat loss varies directly with the temperature difference between the body and its surroundings. Infrared rays are of a

wavelength such that skin color has no effect on the amount of absorption that takes place in contrast to rays in the visible and ultraviolet light ranges, of which 35% are reflected by light skin but only a small proportion are reflected by dark skin. Consequently, in sunlight, dark skin does absorb more heat than light skin. Radiation will, of course, play a minor role in the heat loss story of the submerged diver since radiation is primarily operative in air. Radiation effects would be more important in chamber operations and when the diver surfaces.

Conduction is the direct transfer of heat from the body to a cooler object. This mechanism usually contributes little to heat loss since the object (a chair, for example) would rapidly equilibrate with the body and begin to act as an insulator. It is, however, a major mechanism in diving since the cooler object is water which is continually flowing by the unprotected diver and constantly circulating in small amounts through a wet suit. Since the water absorbs body heat approximately twenty five times faster than air, one can readily imagine the tremendous heat which can be lost by conduction. The situation with a dry suit is only slightly different since the insulating material is mainly air trapped within the suit and suit material. The body heats the air which then remains in place surrounding the skin to provide insulation. Conduction loss occurs at the interface of the dry suit and the water where some heat from the suit is lost to the water.

If the heated air were not held in place by the suit

and were allowed to carry the body's heat away, this would be an example of convection. Convection is when moving air removes heat. Convection loss occurs in conjunction with conduction since heat must be conducted to the air surrounding the body before convection can remove it. With no air movement, the heated air immediately surrounding the body would remain there as insulation just as the heated water in the wet suit remains next to the skin for an 'insulating' effect. With air movement, however, the body can lose as much heat as can be delivered to the body surface. Thus the origin of the wind-chill factor; the faster the air movement, the more potential for heat loss. It is easy to understand why vasoconstriction is so important in maintaining body heat since warm blood would then be kept away from the cooling body surface.

Evaporation/convection is the last mechanism. A small amount of water is constantly being lost from the body surfaces, including the lungs, as water vapor without forming visible water droplets. This water vapor at body temperature is then carried away by convection resulting, normally, in about 25% of the total heat loss. When the body's thermoreceptive centers sense a net heat build-up, one mechanism for its release is by increasing the amount of heat lost by the evaporation/convection route. This is the origin of sweating. Heat produced by exercise is often released by this method.

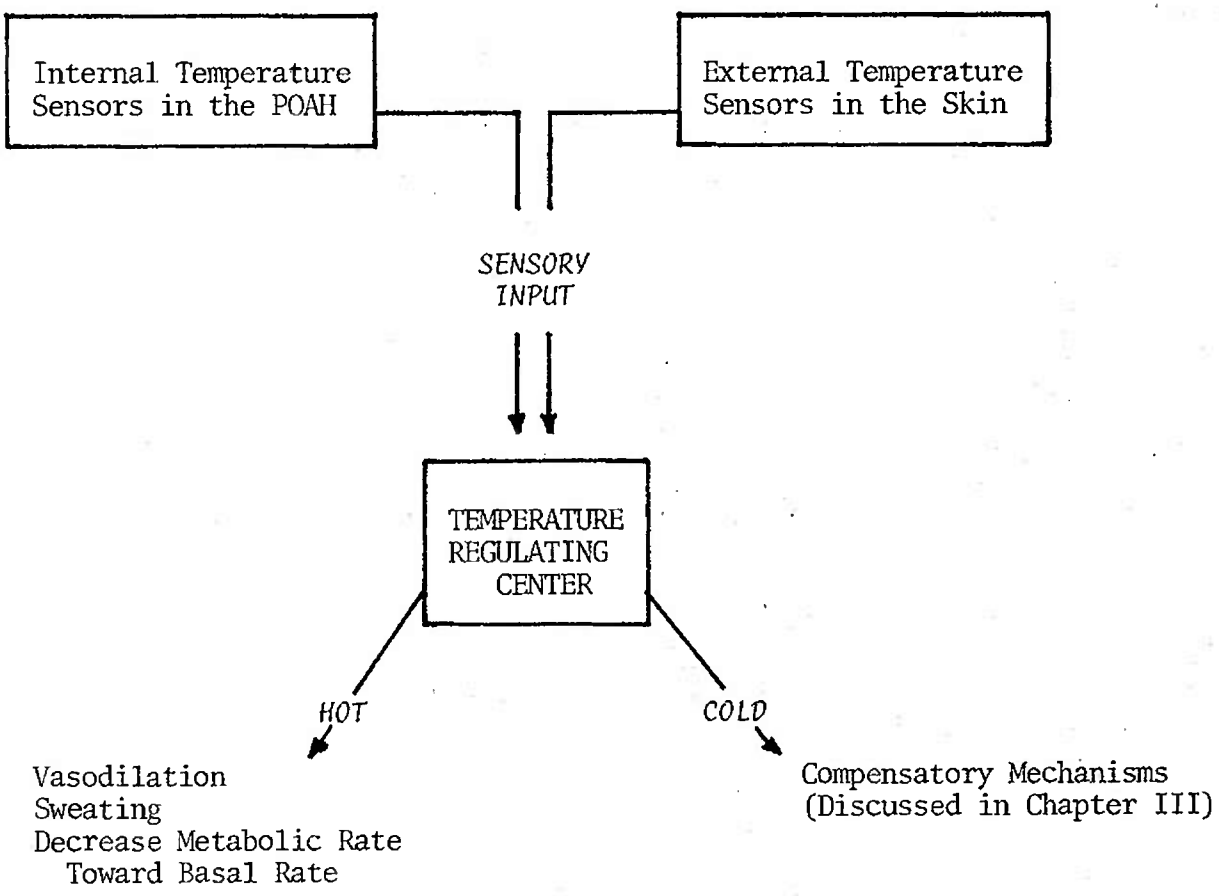
C. Central Regulation of Body Temperature

Body temperature is regulated by nervous feedback mechanisms via the Temperature Regulating Center in conjunction with thermal receptors. The main internal temperature sensors are located in the preoptic anterior hypothalamus (POAH), a part of the brain responsible for many of the unconscious mechanisms sustaining life. These sensors signal the Temperature Regulating Center where deviations from the 'norm' are detected. Secondarily, there are skin temperature receptors for monitoring external heat and cold sensations. Their signals travel to the POAH via the spinal cord where they also interact with some reflexes which will be discussed later.

The fine line of temperature regulation is maintained by the POAH by increasing the metabolic rate thereby increasing heat production when sensors indicate a decline in body temperature. Since there is a basal rate at which metabolism must be maintained and below which cellular functioning would be compromised, the main mechanisms for restoration of homeostasis when body temperature is elevated is to increase evaporative loss and increase blood flow to the skin to increase radiant loss.

This heat gain/heat loss balance is finely controlled under normal conditions and requires only minimal regulatory action. Under conditions of extreme stress, however, these mechanisms must be exaggerated and others invoked to maintain homeostasis as will be discussed in the following section.

AUTHOR'S DIAGRAMATIC REPRESENTATION OF CENTRAL TEMPERATURE CONTROL:



CHAPTER III

PATHOPHYSIOLOGY OF HYPOTHERMIA

The pathophysiology of cold exposure will be discussed in two sections; the first explaining the mechanisms by which the body attempts to produce and conserve heat and the second discussing the effects of progressive chilling on various vital body systems.

A. Heat Production and Conservation

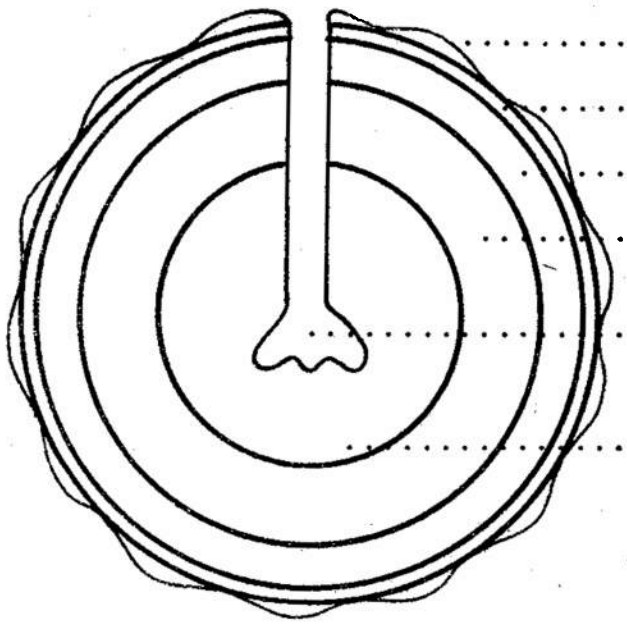
Mechanisms of internal production and conservation of body heat can be categorized as either centrally or peripherally mediated and are listed below to provide an overview before each is discussed individually.

I. Central Mechanisms
    A. Conservative (Sympathetic Nervous System Effects)
        1. vasoconstriction - the most important conservative effect
        2. abolition of sweating
        3. piloerection
    B. Productive
        1. motor effects: shivering - the most important heat source
        2. hormonal (epinephrine, norepinephrine, thyroxine and growth hormone)

II. Peripheral
        1. reset the POAH thermostat
        2. local spinal cord reflexes - controls local vasoconstriction and sweating

III. Protective Outerwear

The body can be thought of as a concentrically layered model where heat is produced by the core and muscle layers and is either lost or conserved by the surrounding fat, skin and clothing layers:



..... clothing layer

..... skin layer with sensory receptors

..... subcutaneous layer (fat)

..... muscle layer

..... lungs - note the portal of entry for cold air to directly affect the core!!

..... the core - represents the vital organs; heart, lungs, brain, kidneys, liver, G-I tract

Each of the body's thermoregulatory mechanisms will affect one of these layers. Centrally, when the preoptic anterior hypothalamus (POAH) receives cold stimuli, conservation of heat is effected by the Sympathetic Nervous System which acts in three ways. First, and most importantly, the peripheral vascular supply is constricted. This vasoconstriction acts to convert the skin and subcutaneous tissues including fat into a functionally relatively avascular insulating layer. This hypoperfused layer conducts heat at a rate of only one-fourth that of normally perfused tissue. An additional effect is to allow the periphery to cool thereby decreasing its metabolic need for oxygen and nutrients. Even though

the metabolic need is decreased, cell activity is still maintained, though at a lower level. As vasoconstriction delivers less oxygen to these tissues, lactic acid is produced as a result of anaerobic metabolism. This lactic acid could be a source of physiologic problems upon rewarming, as will be discussed later. Peripheral vasoconstriction is the most important mechanism of heat conservation. In addition, its action preferentially perfuses the most vital organs: those in the core. This shunting allows the heart and brain to be supplied with nutrients and oxygen at levels nearer to normal than other tissues. It is of interest to note that the body acts to prevent irreversible injury to an extremity by intermittently interposing brief periods of vasodilation to allow limited perfusion to reenter the extremity. This, however, will also allow more heat to be lost when the vessels are dilated - a double-edged sword.

The second Sympathetic Nervous System mechanism of conservation is the abolition of sweating which decreases the evaporative/convective heat loss. The third mechanism is piloerection, a substantial factor in other mammals such as the Alaskan Husky, but inconsequential in man who has only minimal body hair.

The Central Nervous System also acts to increase heat production by both direct (motor) and indirect (hormonal) mechanisms. Under cold stimulation, the POAH causes the motor control center of the brain to increase muscle tone to the point of causing random contractions, the force of which is proportional to the cold stimulation. This is shivering and it can dramatically increase

heat production - for a while. Shivering generates a large quantity of heat but it also consumes a larger amount of energy in so doing and will quickly exhaust the victim. Shivering begins at core temperatures around 97°F and will cease between 90-85°F as the contractions become constant resulting in muscular rigidity. Clinically, one can judge that a man is cold when he begins to shiver, but should be even more concerned when he stops!

Hormonal mechanisms which increase heat production include the cellular effects of epinephrine, norepinephrine, growth hormone and thyroxine. Epinephrine also acts as a peripheral vasoconstrictor assisting the central mechanism. It is of interest that the effects of epinephrine and norepinephrine can be acclimatized; that is, their effects would be more rapid and effective with repeated cold exposures. At approximately 95°F core temperature, the metabolic rate might be increased to six times that of the basal rate.

Peripheral heat maintenance is via two mechanisms. First, through a central feedback mechanism, peripheral temperature receptors act to reset the thermostat located in the Temperature Regulating Center. In other words, if the peripheral receptors are exposed to extreme cold, the central effect might be to 'reset' the TRC to a desired temperature higher than normal. This would prevent the central thermostat from shutting off when the core temperature returns to near normal, even though the body might still be under thermal stress. This action of central thermostat resetting is discussed here under peripheral mechanisms because the stimulus and regulation of the degree of resetting is controlled by the amount of

peripheral stimulus received. Argument can also be made for its inclusion as a central mechanism. It is, in fact, an integration of both peripheral and central controls.

The second peripheral mechanism is via local spinal cord reflexes which decrease sweating and mediate local vasoconstriction in the cold affected area.

The Mammalian Diving Reflex is of interest to studies of drowning and accidental sudden immersion in cold water. It is a complex of reflex bradycardia, apnea and selective perfusion of the heart and brain. Its usefulness is normally restricted to diving mammals which can remain submerged for thirty minutes by virtue of the heat-conserving and metabolic-slowing effects of this reflex. Its only human usefulness is in victims of sudden immersion in cold water. The reflex is initiated when the 'snout' area (nose and mouth) is immersed in cold water. There have been many documented cases of immersions for over an hour where the victims have survived because of the heat preservation and selective sparing of perfusion to the heart and brain. This reflex has also been occasionally used in the treatment of some abnormal heart rhythms in a controlled hospital setting. There is, however, no benefit derived from the Mammalian Diving Reflex in normal diving operations.

B. Effects on Vital Organs

In addition to affecting heat production and conservation, body defenses act on all vital organ systems. Efficiency and effectiveness are increased in essential systems such as the heart,

lungs and brain. Functioning of the other, non-essential, organs is reduced in an effort to conserve energy and heat. In time, however, with progressive heat loss, both normal and compensatory functioning becomes compromised and a precipitous deterioration in vital signs occurs.

Reference will be made to approximate core temperatures at which various affects will be seen. It is important that the reader realize that these are only approximations and can vary by 5-10°F depending on the individual. The effects are extremely variable and the temperatures given are intended to indicate a range rather than a specific number.

Each organ system will be discussed individually:

THE CENTRAL NERVOUS SYSTEM: The effects of cold on the Central Nervous System can closely mimic Type II Decompression Sickness as they cover a wide spectrum of manifestations. Even before the core temperature drops below 95°F (the cut-off for clinical hypothermia), tests of higher functions reveal subtle decrements in arithmetic tasks, logic, word recall, word recognition and dexterity. As chilling continues to near 90°F, cerebral impairment can take the form of analgesia and hallucinations. Hypothermia victims have been recovered in mountainous climes by following a trail of their clothing as they hallucinate a feeling of warmth and well-being, shedding their clothing in their last few minutes of life. Memory impairment is also commonly seen at 90°F. Cold Narcosis, a feeling of well-being, is commonly seen at 85°F By 80°F core temperature, reflexes, including the pupillary light

reflex, are absent and the victim will not understand when spoken to. Below 80°F, obtundation and frank coma are common.

Permanent brain damage is uncommon owing to the relative preservation of blood flow to the brain and to its relatively low metabolic requirement. As long as minimal perfusion is maintained, permanent damage should be avoided. There have, however, been cases of epilepsy and dysphagia reported after recovery from severe hypothermia. These are extremely rare and thought to be due to some greater local ischemia in the areas of the brain responsible for these functions. The explanation for these local effects may be the increased tendency for the cooled, slow-moving blood to form clots and occlude small vessels.

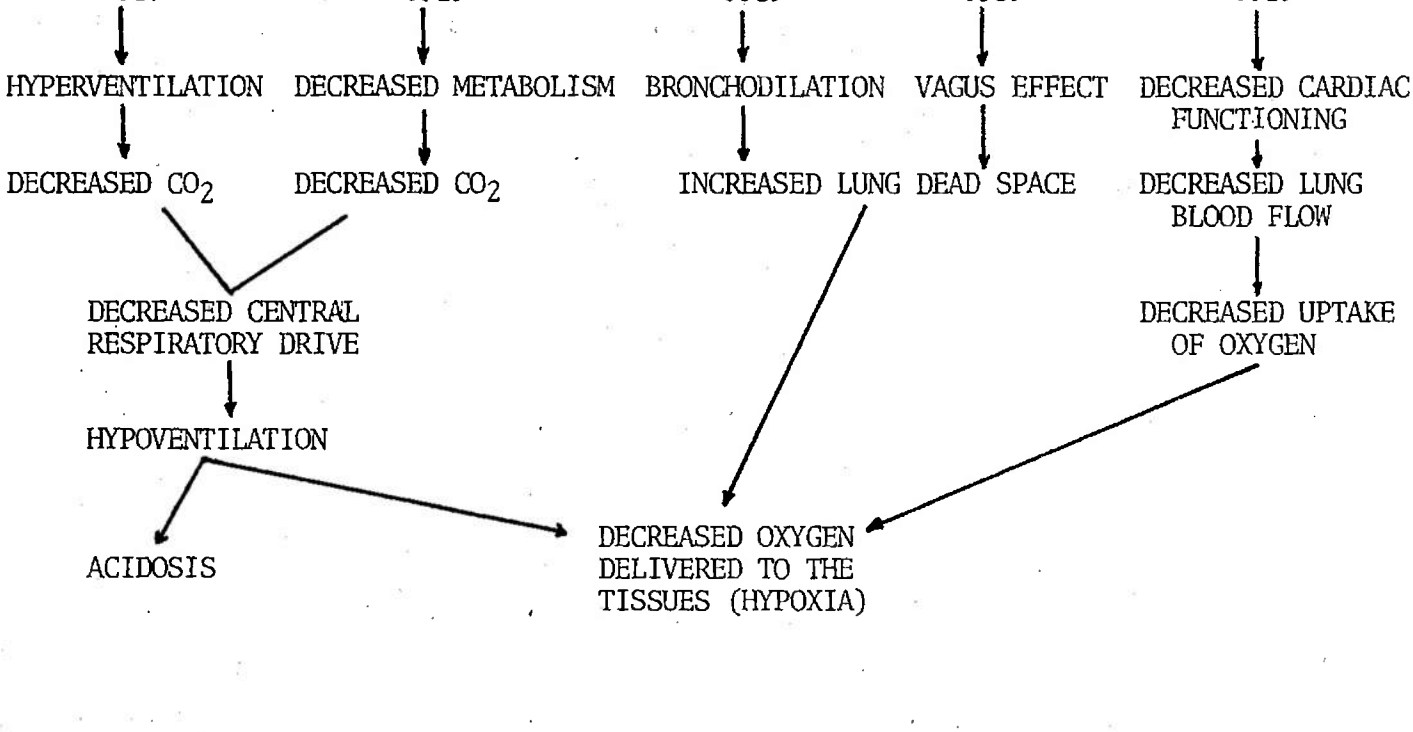
Conduction velocities for peripheral nerves are also reduced; to one-half by 75°F and to one-quarter by 70°F core temperature. Below 70°F, nerve conduction is virtually absent. Remember that the extremities will be colder than the core, hence the effect will be evident peripherally first. Finely coordinated movements of the hands and feet such as manipulation of tools or walking require rapid travel of primary motor and feedback impulses. Clumsiness and difficulty walking are early indications of impaired neural transmission, evidenced after diminution of conduction velocities by only a few percent of normal. These will be important early signs for which the corpsman should be alert.

THE RESPIRATORY SYSTEM: The respiratory response to sudden immersion in cold water is severe and uncontrollable hyperventilation to over ninety liters per minute (as compared with the normal ten

liters per minute) which can eliminate so much carbon dioxide that the respiratory drive is abolished. When hyperventilation is controlled, the victim may not have a stimulus to respiration, thereby inducing apnea. Complicating this is the high probability of inspiring water with these uncontrolled deep breaths. Hyperventilation is also a response when the victim is slowly cooled as in diving operations, but to a much more mild degree and only initially. The carbon dioxide depletion of hyperventilation is coupled with the decreased carbon dioxide production due to tissue cooling and generalized slowing of cellular metabolism. These two mechanisms act insidiously to decrease the central drive to respiration leading to decreases in respiratory frequency and tidal volume. This results in lowered oxygen transported to the tissues and a combined metabolic and respiratory acidosis as metabolic waste products accumulate. Respiration of the severely hypothermic patient must be assisted during resuscitation to avoid this acidosis which can couple with the hypothermia in decreasing the effectiveness of treatment.

Local bronchial cold effect and decreased function of the vagus nerve act together to cause bronchodilation which increases the physiologic dead space (the area of the lungs which contains air but where there is no exchange of gases with the blood). This thereby further reduces the amount of useful new air reaching the alveoli. Add these mechanisms to a decreasing pulmonary blood flow due to the decreasing cardiac output and one can readily appreciate the devastating decline in pulmonary function which occurs.

SCHEME OF COLD-INDUCED PULMONARY PATHOLOGY:



Interestingly, respiratory rhythm appears to be lost only toward the final stages of hypothermia. The rate may decrease to only two or four respirations per minute, but they will be regular. Below a core temperature of 75°F, however, chaotic respirations are the rule.

Direct cold effects on the alveolar and bronchiolar epithelium have been thought to predispose to post-resuscitation infection. Antibiotics, therefore, are often prescribed as part of the post-resuscitation therapy.

THE CARDIOVASCULAR SYSTEM: The initial response to cooling is an increase in blood pressure and heart rate as a result of vasoconstriction and shivering. As shivering ceases and the core temperature continues to drop, the heart rate follows in a linear

fashion. The amount of blood pumped by each stroke (the stroke volume) remains fairly constant, however. This causes a net decrease in cardiac output (the amount of blood pumped each minute) and resultant decrease in tissue perfusion. Even at core temperatures of 70°F, where the cardiac output is reduced to only 20% of normal, the heart and brain are still receiving as much as 40% of their normal supply owing to preferential shunting of the blood supply by peripheral vasoconstriction. The mechanism for this decrease in cardiac performance is slowed electrical conduction within the myocardium, analagous to the slowed conduction within the nervous system discussed earlier. This slowed conduction is evident in the electrocardiogram (EKG) by a prolongation of all portions of the P-QRS-T complex and T-wave inversion. At around 85°F, an extra wave appears within the QRS complex and is known as the J wave.

At core temperatures below 85°F, atrial fibrillation usually occurs spontaneously and ventricular irritability is greatly increased. This makes the ventricles extremely susceptible to fibrillation also, especially between 85°F and 77°F. Fibrillation may occur spontaneously in the ventricles as well or may be triggered by only slight trauma such as movement of the victim. If the heart escapes fibrillation in this period, the heart rate will continue to decrease until all activity ceases. In patients undergoing clinically induced hypothermia for heart surgery, heart rates of four per minute at core temperatures of 52°F have been recorded with complete cardiac standstill occurring at 50°F. These

patients recovered fully from the hypothermia upon rewarming.

The net cardiac effects are a transiently increased blood pressure and heart rate followed by a progressive decrease in blood pressure and heart rate until cardiac failure occurs by either ventricular fibrillation or gradual standstill. Blood pressure is usually not obtainable below around 85°F. The victim may be pulseless and without a blood pressure, however, he may still be resuscitatable due to the preferential perfusing of vital organs and a generalized slowing of cellular metabolism with decreased oxygen and nutrient requirements of all cells.

Of particularly important note is that these cardiac effects are refractory to drug therapy. Drugs have markedly diminished effects in hypothermia and their administration would not only be futile but dangerous because they cannot be eliminated from the body as will be discussed soon.

THE GASTROINTESTINAL SYSTEM: Radiologic studies have demonstrated intestinal paralysis as a result of hypothermia. This response occurs early, around 95°F, as an immediate effect of blood flow redistribution and Sympathetic Nervous System response. This explains why resuscitated hypothermia victims will often complain of abdominal cramping. The gastrointestinal mucosal lining is also a very susceptible target of cold stress. Decreases in mucosal blood flow, as well as stasis and clotting within small vessels, results in hemorrhages and ulcerations. These are often called Wischnevsky ulcers and represent a type of stress ulcer.

This paralytic ileus coupled with vascular compromise and hemorrhage has been linked to the high incidence of pancreatitis found in victims of severe hypothermia. The severity of pancreatitis presents a spectrum from elevated amylase levels recorded in mildly hypothermic patients to severe hemorrhagic pancreatitis seen in the more severe, fatal cases.

The liver, in contrast, is an organ relatively resistant to damage by cooling. Minor changes in liver cells have been demonstrated but are generally reversible. One such change is the rapid (within two hours) depletion of glycogen from liver cells as it is quickly catabolized to glucose for use as a ready emergency energy source for heat production. Just as the metabolic rate of all cells is decreased, the liver is no exception. Because the liver is uniquely tasked with breakdown and elimination of metabolic wastes and toxins, these by-products of metabolism tend to build up. Most notable of these is lactic acid. The most important effects of increased lactic acid are that utilization of oxygen by cells is impaired and myocardial irritability is increased. Catabolism of drugs is likewise impaired. Therefore, if drugs are administered early in the resuscitation effort, before the target organ is able to respond to the drug effects, the drugs will accumulate and remain in their active form. As the resuscitation progresses, the actions of these drugs will be manifest and will be difficult to control until the liver recovers enough to metabolize them. Therefore, the administration of drugs early in hypothermia resuscitation is contraindicated.

THE RENAL SYSTEM: Exposure to immersion temperatures around 60°F is sufficient to elicit a diuresis. This diuresis is partially responsible for the hemoconcentration which will be discussed shortly. The resultant dehydration will require treatment during the resuscitation. Two mechanisms have been proposed for this diuresis. First, decreased reabsorption of the initial plasma filtrate has been proposed due to the decreased metabolic activity of the renal tubular cells responsible for reabsorption. Second, diuresis may be triggered by the false sensation at the kidney of too much blood volume as a result of visceral pooling as peripheral vasoconstriction squeezes blood into the core from the extremities.

The opposite effect, that of oliguria, is seen with prolonged exposure to water temperatures around 85°F. This effect is due to necrosis of tubular cells and occurs primarily in the elderly. Thus, it is of little importance in consideration of diving casualties.

THE ENDOCRINE SYSTEM: Increased activity of almost all endocrine glands has been demonstrated as a result of exposure to cold. Blood levels of cortisol (a steroid) are increased. The thyroid hormone, thyroxine, is secreted in higher amounts resulting in an increased metabolic rate. The adrenal medulla increases secretion of epinephrine and norepinephrine (the former at a rate greater than the latter) in direct proportion to the degree of chilling. The epinephrine and norepinephrine effects are immediate. The other hormonal effects have significant delays from stimulus to secretion and from secretion to effect.

THE BLOOD: Hemoconcentration occurs in response to cold stress via two mechanisms. First, cold diuresis results in a diminished plasma volume as discussed earlier. Second, the permeability of vascular endothelial cells is increased, allowing plasma to leak into extravascular spaces. This increased fluid in the tissues is called edema. The degree of hemoconcentration is directly proportional to the degree of chilling. An increase in hematocrit reflects the degree of hemoconcentration and is correlated with the degree of increased blood flow resistance as a result of the increase in blood viscosity.

The oxyhemoglobin dissociation curve is shifted to the left by the effect of cold alone. In other words, hemoglobin, the oxygen carrying molecule in red blood cells, tends to hold onto oxygen more avidly and not readily allow its use by the tissues. This causes a condition of relative anoxia where there is more oxygen available than can be effectively released to the tissues. This effect is somewhat compensated for by the increasing acidosis which has the opposite effect; that of allowing easier transfer of oxygen from hemoglobin to the tissues. The net effect lies somewhere between the two.

Hyperglycemia occurs during the early phases of hypothermia owing to the glycogen mobilizing effects of the glucocorticoids (steroids) and epinephrine on the liver glycogen stores. Soon, however, liver glycogen stores are depleted and hypoglycemia results. This is followed by increases in plasma-free fatty acid and glycerol levels as fat stores are next to be mobilized as

an energy source. This is caused by the lipolytic (ability to break down fat) effect of epinephrine and norepinephrine on fat cells. Mild ketosis results as with all metabolism dependent on fat as an energy source. The effects on lipid metabolism are not as immediate as the effects on glycogen stores.

Electrolyte disturbances are seen in severe hypothermia as water and sodium are driven into cells and potassium is forced out. This results in tissue edema, mild diminution of serum sodium (compensated for by hemoconcentration and dehydration) and increased serum potassium levels.

Clotting tends to occur more readily within the small arterioles as the viscosity increases, the platelet count increases, the rate of blood flow decreases and thrombogenic compounds such as histamine and various kinins are released by direct action of cold on the cells. As microclots form, occlusion of small vessels results in damage to vital organs such as the brain (stroke), heart (infarction of myocardial tissue; heart attack) and the gastrointestinal tract (stress ulcers). These effects are usually not seen until the core temperature slips below 80°F.

CHAPTER IV

CLINICAL PRESENTATION

The clinical presentation may be conveniently divided into the subjective and the objective findings. Subjective findings refer to the symptomatology; what the patient feels and describes. Objective findings (signs) refer to those phenomena observed by the examiner.

The following presentation of these signs and symptoms, as correlated with core temperature, is intended only as a guide. The corpsman must be aware that there is tremendous variability in individual responses. The variables, alluded to previously, of nutrition, conditioning, body fat content and habitus, etc., as well as the basic individual variations in body constitution (differing Basal Metabolic Rates, for example) will afford more or less of a functional decrement to one individual at a core temperature which would result in a different response for another It is unknown, for example, why ventricular fibrillation would intervene in some victims as their terminal event at around 77°F, yet not occur for others who expire of gradual cardiac standstill at a much lower core temperature. The rule is to guide therapy by the clinical presentation of the patient, not by the core temperature alone.

A. Subjective Findings

The onset of hypothermia is much like that of falling

asleep; one doesn't realize when he slips from consciousness into the sleeping state unless he thinks about it. One must also be thinking about hypothermia to recognize it. With continuous exposure, the effects of hypothermia are insidious and eventually reach a point when, as all the body's emergency heat production and conservation measures have failed, the course is precipitously downhill.

The only subjective effects of gradual core cooling are concerned with the Central Nervous System and the local effects of cold on muscle strength and efficiency. The victim will naturally 'feel cold'. The first subjective effects will be decreased dexterity and coordination due to local effects of cold on extremity muscles. One will almost immediately notice manual functioning becoming slowed and coordination diminishing; he will become clumsy and inefficient. Nerve conduction is also decreased, contributing to loss of dexterity and sensation. There is also diminished proprioception (feeling of position and angle of joints).

These effects occur as the extremities are cooled, yet the core temperature has not yet begun to decline. As core temperature begins to drop, the hypothalamus begins to turn on the heat production and conservation mechanisms. One of the first methods of heat production is to increase skeletal muscle tone thereby increasing its metabolic rate. All skeletal muscles are affected and, as the temperature continues to decline, the increased tone becomes shivering, which can be violent. Shivering begins at about 97°F and is maximal at around a 95°F core temperature.

One result of the increased tone is bilateral temporal headaches due to the contraction of the temporalis muscles. The local effects of cold on extremity muscles is compounded by the heat conservation mechanism of vasoconstriction. This diminishes blood supply and, as shivering is established, the tissues become anoxic, accumulating lactic acid thereby further diminishing muscle efficiency.

The victim usually remains conscious and oriented at core temperatures as low as 92°F. Below this temperature is generally considered 'severe hypothermia' and diminished cerebral functioning becomes increasingly evident. The victim's consciousness begins to cloud, his orientation fades. Muscle rigidity is constantly increasing, making voluntary motion difficult. Consciousness declines as stupor and, eventually, coma intervene at core temperatures below 85°F.

The corpsman must be thinking of hypothermia to recognize it. Obvious symptoms of severe hypothermia will be preceded by the subtle symptoms of increasing muscle tone, clouded sensorium and diminished coordination. It should be these softer manifestations of which the corpsman should be suspicious.

B. Objective Findings

The objective findings of hypothermia are not as subtle as are the subjective. A complete physical examination is essential in the diagnosing and assessing of hypothermia after it is suspected by the manifestation of the softer evidence discussed above. Determination of the core temperature is very important.

Oral and axillary temperatures are unsatisfactory since they are too peripheral; not close enough to the vital organs. Rectal and esophageal temperatures are the best methods. Rectal temperatures very closely approximate the core temperature owing to the rich blood supply of the area. Esophageal temperature is the best since it measures the core temperature closest to the heart and lungs. This is, however, not practical with the facilities normally available to the corpsman in the field. Special thermometers must be utilized which record temperatures lower than the 94°F of the usual clinical thermometer. A systematic approach to the physical examination should be the routine and the following discussion will be presented systematically as the core temperature declines.

The earliest signs of hypothermia are, by no means, pathognomonic. The cool extremities with a barely palpable pulse and increased muscle tone will be found in anyone exposed to significant cold. It is the combination of the above subjective findings and the objective findings that will make the diagnosis apparent. One would see cool, dry extremities lacking coordination and strength, gait is affected, speech is slowed and there is mental clouding. Patients are, however, usually conscious and responsive with a normal blood pressure until their core temperature reaches 92°F (the level of clinically severe hypothermia) and should be shivering initially.

As the core temperature declines to between 92°F and 85°F, one's consciousness becomes increasingly dulled, coordination

increasingly faulty, speech increasingly slurred. Closer to 85°F, the level of consciousness is markedly affected resulting in stupor. The pupils dilate but remain reactive to light. Respirations are diminished but are still regular and spontaneous. Pulse, although difficult to obtain due to the peripheral vasoconstriction, is slowed yet also remains regular. Gastric motility is absent. By this time, shivering has ceased and muscle rigidity remains, thereby dramatically decreasing voluntary motion.

Between 85°F and 80°F, the victim is seldom conscious but should be arousable. Respirations and pulse are further slowed. Myocardial irritability is at its greatest near 80°F with arrhythmias and fibrillation resulting from very slight irritation. Fibrillation is often the terminal event in this temperature range. At 80°F, consciousness is lost, there is no motion, reflexes are absent (including deep tendon and the pupillary light reflexes). Pulse, respiratory rate and blood pressure are unobtainable. In short, the victim appears to be dead. Do not lose hope at this point, however. The victim's vital tissues may be protected by the decreased metabolic rate and the low oxygen requirement resulting from the compensatory mechanisms of hypothermia. He must be warmed to assess his condition. *The CARDINAL RULE of hypothermia is that one is not dead until he is WARM and dead!!!*

Below a core temperature of 80°F, most vital functions are at the lowest level compatible with life. Further deterioration in respiratory or cardiovascular function will often result in

permanent damage. Ventricular fibrillation often occurs spontaneously. Respiration is depressed until all breathing stops. Damage to lung tissue by direct effects of cold results in pulmonary edema further hindering gas exchange and making the resuscitation efforts more difficult. Around 70°F, the heart will usually slowly stop spontaneously if fibrillation has not intervened as the terminal event. It is important to note that even if the victim is conscious, cardiac irregularities can be present and irritability increased. Indeed, nonpathologic irregularities occur in the health normothermic individual.

The corpsman will seldom see the advanced signs of hypothermia. Instead, the subtle initial signs will be the only clues. The hypothermia which will occur in normal Navy diving operations will only be of the mild type evidenced by deficits in mentation and dexterity. The more severe hypothermia victims will be seen as the result of accidents; the drunk on a park bench in January, the widow living alone in an unheated flat, the child who doesn't reach the other side of the thin ice or the mountaineer who is caught unprepared for a chilling spring storm. Prevention is the answer, treatment is only second best.

SIGNS OF HYPOTHERMIA AS APPROXIMATELY RELATED TO CORE TEMPERATURE

°C	°F	
37.6	99.6	'Normal' Rectal Temperature
37	98.6	'Normal' Oral Temperature
36	96.8	Metabolic Rate Increases - trying to overcome heat losses
35	95.0	Shivering Maximum
34	93.2	Victim still conscious with normal blood pressure
33	91.4	*SEVERE HYPOTHERMIA BELOW THIS LEVEL*
32	89.6	Consciousness becomes clouded, pupils dilate but remain reactive to light, blood pressure becomes difficult to obtain.
31	87.8	
30	86.0	Progressive loss of consciousness, muscular rigidity increased, pulse and blood pressure very difficult to obtain.
29	85.2	
28	82.4	Ventricular fibrillation may develop if heart irritated, cardiac arrhythmias develop, atrial fibrillation occurs spontaneously.
27	80.6	
26	78.8	Victim seldom conscious
25	77.0	Ventricular fibrillation may occur spontaneously
24	75.2	Pulmonary edema develops
23	73.4	
22	71.6	Maximum risk of ventricular fibrillation
21	69.8	
20	68.0	Cardiac Standstill
19	66.2	
18	64.4	
17	62.6	Flat-line Electroencephalogram (EEG)
16	60.8	
15	59.0	Lowest accidental hypothermia victim to recover
9	48.2	Lowest artificially cooled hypothermia patient to recover

CHAPTER V

TREATMENT

The topic of treatment will be discussed in two parts; the first dealing with first aid and general management of the hypothermia victim and the second dealing with specific rewarming techniques.

A. First Aid and General Management

The time-honored cardinal rules of first aid should be observed in the treatment of hypothermia as well. First, there is usually no great rush since the progression of hypothermia is slow and the victim's fate will usually not be greatly altered form one second to the next unless cardiac standstill has intervened. Indeed, if one were overly zealous in his first aid, ventricular fibrillation could be triggered by the well-meaning first aider. Recall the increasing myocardial irritability!!

The ABC's of standard first aid apply; ensure an established airway, support adequate breathing and maintain circulation. If cardiac standstill has occurred, cardio-pulmonary resuscitation (CPR) should be initiated with chest compressions at one-half the normal rate if the core temperature is below 92°F. The secondary principles of first aid should then apply while carefully moving the victim from the environment and beginning the rewarming procedure. These secondary principles involve treatment of bleeding, stablizing of fractures, etc. Keep in mind that sophisticated

hospital treatment will be necessary for any victim whose core temperature is less than 92°F and that it will not be the responsibility of the corpsman to totally manage the hypothermic victim.

Treatment modes will depend on the victim's degree of hypothermia. A mildly hypothermic patient is one whose core temperature is greater than 94°F and who is conscious and shivering. These patients will have retained the ability to rewarm themselves if removed from the environment. They are in no serious danger and will not require hospitalization. Their treatment will consist of simple rewarming methods and supportive care. These are the patients who the corpsman will usually be seeing since the mental and physical functional decrements will be noticed before serious hypothermia is encountered. Proper supervision will result in suspicion concerning diminished performance and subsequent removal from the offending environment. The mildly hypothermic victim should remain out of the cooling envirnment until *completely* rewarmed.

Depending on the available facilities, the victim of severe hypothermia should be removed from the cooling environment, monitored with an EKG and core thermometer, hydrated with (warmed) intravenous fluids, given a glucose infusion to compensate for the hypoglycemia and core rewarming should be initiated. As alluded to previously, the cardiac arrhythmias associated with hypothermia are unresponsive to pharmacologic treatment and may be converted to more serious arrhythmias with the intervention of countershock therapy or attempts at defibrillation. Therefore, drug therapy and countershock or defibrillation should be reserved until the core temperature is elevated above 90°F. Adequate

circulation can be maintained with proper cardiopulmonary resuscitation techniques even while the heart is in fibrillation.

The severely hypothermic patient will obviously require advanced medical attention beyond the capabilities of a small dispensary. He will require a medical center and the attention of physicians with specific knowledge regarding the special aspects of hypothermia treatment. Hence, the above short summary of treatment of severe hypothermia was not intended as a guide to therapy, but only as an overview so that the corpsman is aware of the general principles of initial management of severe hypothermia. In actual treatment, all of the previously discussed effects of the cold must be considered and treated by medical personnel.

The corpsman's thoughts should be first directed toward basic maintenance of body functions through observation of the ABC's of first aid. Secondly, attention should be focused on rewarming the victim.

B. Rewarming Methods

There has been a great deal of discussion in the scientific literature regarding the proper method of rewarming. Two general categories have emerged; central rewarming and whole body rewarming. Whichever mode is selected, the general rules of hypothermia treatment should be observed:

1. *Rewarm the victim only when it can be done properly.*
2. *Handle the victim carefully.*
3. *Prevent further heat loss.*
4. *Do not rewarm too quickly.*
5. *REWARM THE CORE FIRST.*

Generally speaking, the dividing line between these two categories of therapy should center around the physical findings as an indicator of the severity of hypothermia. If the victim is still conscious and shivering, then one can be safe in assuming that he is only mildly hypothermic and can be rewarmed by any of the whole body methods. He will not be severely acidotic nor prone to myocardial irregularities. Additionally, the body will have retained its ability to rewarm itself if removed from the cooling environment.

Whole body rewarming methods include the following:

1. Removal of the victim from the offending environment.
2. Application of blankets and other warming and insulating materials.
3. Application of moderate external heat to the entire body with a warm shower or by sitting in front of a stove and also placing warm packs on the 'pressure points' in an attempt to directly warm the blood.
4. Ingestion of warm fluids such as hot chocolate (caffeinated beverages should be avoided due to their vasoconstrictive effects).
5. Huddling one sparsely-clothed body with another can be used with only limited success, but better than nothing.

Application of any of these methods will see immediate improvement in the symptomatology of mild hypothermia. The state of consciousness will improve, shivering will abate, dexterity will improve and sweating will present as the end point of rewarming.

In contrast, the severely hypothermic victim would be made precipitously worse by these methods. As mentioned earlier, the peripheral vasoconstriction and increased muscular activity of

shivering results in peripheral chilling to a greater degree than that of the core and high levels of lactic acid accumulate as the muscles contract anaerobically. Consequently, if the periphery were rewarmed before the core, there would be a sudden and massive influx of lactic acid and cold blood. This would worsen the already present metabolic acidosis and would further drop the core temperature as the cold blood mixed with the warmer core blood. This drop in core temperature is called the *afterdrop*. Afterdrop can be fatal as it lowers the core temperature to a more dangerous level than before and by insulting the already irritable myocardium with a bolus of cold blood. The rule in hypothermia (where consciousness is severely blunted and shivering has abated) is to rewarm the core actively and allow the periphery to rewarm passively at its own rate. In this manner, only small amounts of cold, acidotic blood will leak into the core at a time and can be more readily managed by the heart, lungs and liver (which metabolizes the lactic acid) whose function will already hopefully be returning to near normal by the core rewarming methods before the periphery begins to warm.

In contrast to the whole body rewarming methods which are readily accomplished with little expertise, only the first two methods of core rewarming are easily performed outside the hospital environment. These should be initiated in the severely hypothermic victim while transport to a hospital is arranged. Remember, severe hypothermia requires hospitalization to treat the related problems adequately.

In principle, core rewarming is intended to; first, minimize the time the victim is hypothermic; second, quickly revitalize the weakened and irritable myocardium; third, avert the afterdrop phenomenon by overwhelming the temperature gradients within the victim's body with a massive infusion of heat applied to the core. A thumb rule in core rewarming is that a rate of 1°F per hour rise in core temperature is about optimum (although some controversy exists, this is the most widely accepted rate). This seems to be the maximum rate at which a good clinical result is obtained with the least probability of complications from too rapid rewarming.

One of the best methods of core rewarming is immersion of the trunk only in a hot water bath. The water should be about 105°F, a few degrees hotter would result in damage to the skin already susceptible to injury from being traumatized by the effects of hypothermia. This method has been shown to be one of the best because it allows for core rewarming while excluding the extremities, leaving the head and extremities free for inhalation and intravenous therapies to be initiated concomitantly. It is also easy and inexpensive. The only contraindications are trauma involving open wounds to the abdomen or chest requiring chest tubes and, of course, if CPR is indicated. Disadvantages are the inaccessibility of the chest for CPR, difficulty monitoring the heart and lungs under water and, although it is a relatively easy method, if a suitable basin is not available, it can be very awkward. Core temperatures should be frequently monitored with a remote probe if possible to obviate the need to constantly manipulate the patient.

The second method is by inhalation of warmed, moist air or oxygen. Early efforts in this area were made using hospital anaesthesia equipment and, later, hot water baths to heat the oxygen. Now a small inhalation apparatus is available which can be carried into the field. It moistens and warms oxygen to an optimum temperature of 110-112°F. The advantages of inhalation therapy are that it allows for selective warming of the heart and lungs, the equipment is simple, inexpensive and easy to operate and transport and requires no external power source. A contraindication is severe trauma to the face although this can be circumvented by performing a tracheotomy or by intubation. A disadvantage is that the patient must have spontaneous respirations although this, too, can be remedied by tracheotomy or intubation. As before, adequate monitoring is required. The inhalation method has been shown to be about as effective as trunk immersion, requiring only the equipment and a little training in its use.

Peritoneal irrigation is a hospital procedure which involves placing a catheter into the peritoneal cavity and instilling warmed (to about 105°F) fluids. It is very similar to peritoneal dialysis which uses the large surface area of the peritoneal cavity for removing waste products from the blood like an artificial kidney. This large surface area provides an opportunity for rapid core rewarming as well. Advantages are that, in trained hands, the procedure is simple and safe, it applies heat centrally, it allows for dialysis of a toxin or drug which might be present in the victim's circulation, it allows for correction of electrolyte

imbalances when the proper irrigation fluids are utilized, it does not interfere with concurrent treatment and it can be used in conjunction with inhalation rewarming methods. A contraindication is severe trauma to the abdomen. Under this circumstance, warm irrigation during surgery would substitute. Its disadvantages are the expertise required to perform the procedure and the requirement for laboratory facilities for careful monitoring of the electrolytes.

The above three methods are the most practical and widely used core rewarming methods. The following are mentioned for the interest of the reader.

Gastric and colonic balloons have been utilized by circulating warm solutions through them. Their advantages are the simplicity and ease of operation since no particular expertise is required nor careful selection of the irrigant solution. Disadvantages are that the balloons are seldom available, their surface is smooth and, therefore, does not affect nearly as large a surface area as the latter three methods, if the balloons break their contents would spill into the respective body cavities potentially creating osmotic or electrolyte imbalances if absorbed and, lastly, the area of the heart and lungs is only secondarily warmed.

Colonic enemas have been employed with success. Their advantages are the ease and simplicity of instilling a warmed enema and that electrolyte imbalances can be partially corrected with proper selection of the fluid to be instilled. Disadvantages are that it does not allow rectal temperature monitoring, it does not allow direct warming of the heart and lungs and once the enema is instilled, recovery may be difficult if complications should

arise requiring its evacuation.

Extracorporeal blood rewarming is a very effective method but requires very sophisticated equipment and a specially trained team of surgeons and technicians available only at the most advanced hospital centers. It also requires the use of anticoagulants which might be contraindicated in the presence of trauma.

Diathermy, or microwave therapy, is sometimes used to heat the core preferentially. Diathermy is mainly utilized in the treatment of muscle spasm because of its deep heating action. It is, however, fraught with hazards in the treatment of hypothermia and is mentioned here only in passing.

In summary, the principles of hypothermia treatment are simple. First, remember the ABC'c of first aid; ensure an established airway, support adequate breathing and maintain circulation. If CPR is required, perform it at one-half the normal rate if the core temperature is below 92°F. Secondly, treat active bleeding, stablize fractures and treat life-threatening trauma. Third, determine the level of hypothermia. Next, keep the victim hydrated and give glucose if it's available. Monitor with an EKG and rectal thermometer if possible in severe hypothermia. After assessing the severity of hypothermia, select an appropriate rewarming method and obtain sophisticated medical assistance if necessary.

Remember - be gentle! The cooled myocardium is prone to fibrillation if insulted. Recall that drugs have no place in early resuscitation efforts since they have no effect on the hypothermic heart and would only accumulate to have their effects manifest later when they might not be desired.

SUMMARY OF HYPOTHERMIA TREATMENT MODALITIES

MILD HYPOTHERMIA:

1. Remove the victim from the chilling environment.
2. Apply blankets and other external warming and insulating materials.
3. Apply external heat in the form of warm showers, radiant heat, hot packs to 'pressure points'.
4. Ingestion of warm fluids, avoiding caffeine.
5. Huddle if no other means is available.

SEVERE HYPOTHERMIA:

	Advantages	Contraindications	Disadvantages
1. Trunk Immersion	Readily available Excellent result Allows preferential core rewarming	Trauma with open wounds CPR necessary	Chest inaccessible for CPR Difficult to monitor heart and lungs Must have large basin or tub
2. Inhalation	Leaves chest available for CPR Very effective Selective rewarming of heart and lungs Curtails respiratory heat loss Can be combined with #1 Simple equipment which is inexpensive, easy to transport and requires no external power	Severe trauma to the face (although tracheotomy or intubation can be performed)	Patient must have spontaneous respiratio (although tracheotomy or intubation can be performed)

3. Peritoneal Irrigation	Preferential core rewarming Simple and safe in trained hands Allows dialysis of toxins and metabolic wastes Does not interfere with concurrent treatments Can be used with #2	Severe abdominal trauma	Requires some expertise and laboratory facilities
4. Gastric & Colonic Balloons	Simple and easy to use No special irrigant needed	Severe abdominal trauma	Balloons seldom available Balloon surface area small Balloon may break
5. Colonic Enema	Simple and easy Electrolyte corrections can be made	Severe abdominal trauma	Cannot monitor rectal temperature Does not warm the heart and lungs well
6. Extracorporeal Circulation	Warms blood very effectively Excellent control of rewarming rate	Any trauma or other factor which would obviate the use of anticoagulants	Requires highly skilled personnel and very sophisticated and expensive equipment
7. Diathermy	Warms deeply	Many	Great risk of tissue damage

CHAPTER VI

PREVENTION

The key to management of the hypothermia problem is prevention. There is no magic to it, just common sense; first, keep warm. Second, if you must enter a cold environment, be properly prepared. Third, it you can do neither of the first two or they are ineffective, then conserve body heat by a proven method.

Keeping warm is strictly a common sense step. Be adequately clothed for the weather; if it's raining, wear waterproof gear, if it's cold, wear clothing in layers. Layering has been shown to be the most effective method of wearing a given weight of clothing. That is, wear a T-shirt, long underwear, a regular outer shirt, insulating vest, thick outer coat and then a water repellent garment. This is much better that just a shirt and one heavy coat. Insulation effect is based on trapped air as the insulator and the more layers worn, the more air is trapped. Wool has been shown to be the best clothing material because of its superior air-trapping and its ability to retain its insulating properties when wet. Of important note is the need for head protection. Because the scalp vessels do not significantly vasoconstrict, the heat loss from the head can be as high as 80% of the total heat loss. Hence, wear a wool watch cap, hood or balaclava.

Expose as few people to the cold as possible. Personnel not directly involved in diving operations should be inside where

it's warm. This is especially true for the divers who will be exposed to exceptional stresses when they enter the water. After exposure to the cold, be sure to warm up *completely*. It is counter-productive to warm the extremities so that they just feel good, then return to the cold. This has two effects; first, the cold extremities would vasodilate allowing the cool muscle to reduce the temperature of the increased blood flowing through them, thereby secondarily cooling the core. Secondly, this vasodilation will cause heat loss from the extremities at a much greater rate when returning to the cold environment. If one must return to the cold environment in a short time, it is better to remain cold and peripherally vasoconstricted than to warm up partially because of these deleterious effects of vasodilation and reexposure.

Protective clothing not only pertains to the topside personnel, but also to the divers. Generally, there are three types of exposure suits available; the free-flowing hot water suit, the wet suit and the dry suit. The free-flowing hot water suit requires extensive shipboard facilities and is intended for use in tethered systems. It is loosely fitting and constructed of a closed-cell neoprene material which is open at the ankles and wrists. This allows warmed water to circulate freely through. This system is only of value in large scale diving operations. Skin maceration due to constant immersion in warm water is often the time-limiting factor.

The wet suit is a skin-tight garment constructed of 3/16" or 1/4" closed cell neoprene rubber. It is designed to fit snugly

enough that outside water is prohibited from circulating next to the skin. The insulating ability of this system is based on the insulating ability of the gas trapped in the closed cell foam construction of the neoprene. The gas, however, is compressed (according to Boyle's Law) when descending so that at four atmospheres of pressure (approximately 100 feet), the insulating ability is reduced to only 25% that at the surface. This system is, therefore, quite ineffective at significant depths.

The dry suit, in contrast, completely isolates the diver from direct contact with the water. It may be made of loosely fitting closed cell neoprene (the Unisuit) or a tough snagproof rubber or rubberized canvas (the MK V Deep Sea Diving Dress). Provision is made to add gas to the suit as the diver descends to keep the volume of the suit constant. The insulating value of the suit is a function of the type of undergarment worn, its thickness and the amount of gas trapped in the suit. Therefore, the diver should wear wool diving underwear, socks and a wool watch cap. Air is a better insulator than helium. Thus, when diving HeO_2, there is a much greater potential for heat loss. The HeO_2 diver will become hypothermic at a much greater rate than with air.

Supplemental systems are being developed to allow warming of a layer of water held in a tube suit between the diver and the dry suit itself. This would decrease the temperature gradient between the diver and the suit. An attempt is also made to heat the diver's inspired air to lessen the respiratory heat loss.

This is, however, extremely difficult considering the long hose necessary to reach the diver at depth. Strict temperature control must be maintained when changing depth since, when ascending, there would be less cooling of the hot air by water and the diver could suffer respiratory tract burns.

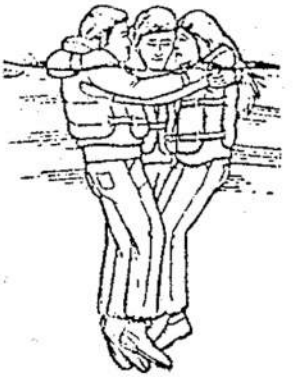
Adequate surface intervals cannot be stressed enough. Thorough rewarming is the rule and a guideline is that one is adequately rewarmed when sweating begins. Good diet and fluid intake should be adhered to. Alcohol abstinence is imperative since alcohol is a vasodilator. Cigarettes and coffee or tea should be avoided because nicotine and caffeine are vasoconstrictors.

Finally, if all else fails or the effects of environmental cold surpass the body's efforts to maintain thermal homeostasis in the water, there are additional methods of heat conservation that are more accurately termed survival rather than preventive measures. Always wear a life jacket or an inflatable vest. Since the greatest amount of potential heat loss is through the head, keeping it out of the water is essential. Drownproofing is a very unsatisfactory procedure for this reason. Without flotation, valuable energy must be expended to maintain oneself on the surface. With flotation, two methods of heat conservation are recommended. If you're alone, use the H.E.L.P. (Heat Escape Lessening Position) as shown in the following picture. The legs are crossed and bent with the knees held toward the chin. The arms are wrapped around the legs or a flotation device. If possible, the hands should be placed in the axillae. This posture lessens

the body surface area in contact with the chilling water. If accompanied, use the Huddle position. This is done by remaining vertical in the water and closely holding your companions. These methods are the best for maintaining heat when immersed.

Remember, survival time in the water depends largely on two factors; the temperature of the water and the behavior of the victim. Prevention of hypothermia is very simple. In fact, it's common sense! Adherence to these few principles will not only decrease morbidity from hypothermia, but also increase the enjoyment of working in the cold environment.

The H.E.L.P. Position



The Huddle Position

SECTION TWO

CORPSMAN REFERENCE HANDOUT

HYPOTHERMIA

I. INTRODUCTION

Hypothermia connotes the abnormal lowering of body core temperature (the internal temperature of the body; that of the core organs) the effects of which are in a gradient from only a mild decrease in motor and cognitive functions to the severe reactions of cardiac and respiratory failure. Hypothermia becomes important to the corpsman who will accompany personnel into the thermally stressful environments of cold weather and cold water.

This presentation is designed to provide the corpsman with a basic understanding of the body's normal thermoregulatory mechanisms, their methods of adaptation to cold stress and the clinical syndrome which results. Knowledge of the basic pathophysiology will allow the corpsman to interpret the subtle objective indicators of hypothermia and to recognize the vague subjective indicators as voiced by the victim. This background will facilitate prompt treatment and relief of a potentially dangerous malady.

II. NORMAL THERMOREGULATORY MECHANISMS

Maintenance of a normally stable body core temperature is a carefully controlled balance between heat production and heat loss. Fever results from excess heat production without concomitant loss, whereas hypothermia results from the inability of heat production to keep pace with heat loss.

Heat is produced at a variable rate for each individual. This rate is affected by the actions of the Sympathetic Nervous System, exercise, hormonal influences (especially epinephrine and thyroxine) and the direct effects of temperature on cellular metabolism. The greatest effects are those of exercise and hormones, each able to increase the metabolic rate a hundred-fold. The effect of hormones is the slower of the two, involving a lag period between stimulus, secretion and effect. Exercise as manifested by shivering is an immediate effect and at a level such that a twenty-fold increase in heat production can be sustained. This form of heat production is, however, very costly in terms of total body energy resources. The energy, in the form of glucose, which is required to sustain shivering will rapidly deplete the body's stores of glucose requiring the body to then obtain energy from fat, a less efficient and slower process.

Heat is lost via four mechanisms. The first is radiation which usually accounts for about 60% of the total heat loss. Heat is

transferred to the surroundings in the form of infrared radiation. The warmer objects will transfer heat to the colder. This mechanism is operative in air, less so when protected by clothing and only minimally when immersed in water.

Conduction is the major mechanism of heat loss in water. Heat loss by conduction is the transfer of heat from one object or medium to another by direct contact. A conducting object or medium which remains stationary will act as an insulator when a temperature equilibrium is achieved. If the object or medium moves, however, it takes the conducted heat away. This is the active mechanism by which heat is lost in diving. Conduction effect can, therefore, be obviated somewhat by not allowing the heated water surrounding the body to move away.

Heat is also conducted to air and, when the air moves away, the third mechanism of heat loss is evident: conduction/convection. By convection, the heated, insulating air is removed from near the skin and replaced with cooler air into which more heat is lost. This effect can be minimized by retaining the warmed air near the skin. Indeed, this warmed air can be used as an insulator. This is the principle of using many air-trapping layers of clothing when exposed to cold air and of the air-filled dry suits used for cold water diving.

Evaporation of surface water into water vapor which is then carried away from the body by convection is the fourth mechanism of heat loss. Normally accounting for 25% of all heat loss, this mechanism is invoked for rapid loss of excess heat by increasing the amount of water evaporated until sweating results.

Thermal homeostasis is a balance between production and these four heat dissipating mechanisms. This balance is mediated by the Central Nervous System through an area of the brain known as the Temperature Regulating Center. The Temperature Regulating Center receives sensory input from monitors within the brain which sense internal temperature (that is, the temperature of the blood) and also from skin receptors which sense peripheral temperature. The Temperature Regulating Center then determines the proper mix of heat producing and heat losing mechanisms to maintain a constant body core temperature.

III. Pathophysiology of Hypothermia

A. Heat Production/Conservation Mechanisms

The body can be graphically viewed as a concentrically layered model with the heat producing core and muscle layers in the middle and the variably heat losing or conserving subcutaneous and skin layers on the outside. One additional layer may be added to the outside to represent clothing.

With exposure to a cold stimulus, mechanisms of both heat production and conservation are adjusted. These effects can be categorized as either central or peripheral. The central effects can be further delineated as conservative or productive. The Sympathetic Nervous System mediates the mechanism of centrally controlled heat conservation in three ways. First and most importantly, the peripheral vascular supply is constricted. This allows blood to be shunted from the cold periphery to the vital core organs (heart, brain, lungs) thereby preserving core functions. A detrimental effect of vasoconstriction is that it markedly reduces blood supply to the extremities requiring the shivering muscles to metabolize anaerobically thereby producing large amounts of lactic acid. Peripheral nerves also become anaerobic resulting in decreased function which tends to further reduce motor function and diminish sensory perception. The second Sympathetic Nervous System effect is to abolish sweating. Piloerection, the third effect, is not a significant mechanism in humans.

The central effects resulting in increased heat production can be divided into motor and hormonal effects. Motor effects are evident as shivering which is the mechanism of rapid heat production. Shivering will eventually cease as the core temperature drops and the increased muscle tone which produced the shivering will then result in muscle rigidity. Hormonal effects include the actions of thyroxine, epinephrine, norepinephrine and some minor hormones to increase cellular metabolism.

Peripheral effects involve feedback of sensory nerves in the periphery with the Central Nervous System or with other peripheral nerves. First, continued cold stimulation results in a resetting of the internal thermostat located in the Temperature Regulating Center. This assures a continued heat production drive even when the body temperature nears normal. Secondly, local reflexes also cause local vasoconstriction. Thus, a single cold extremity or part of an extremity can be vasoconstricted without affecting the entire body.

A third category of temperature regulation which is neither central nor peripheral is the use of clothing as an insulating layer. Properly layered clothing or an appropriate immersion suit can significantly improve heat retention. It is especially important to protect the head since the vascular supply to the scalp does not readily constrict. The warmed blood supplying the scalp will, therefore, be always vulnerable to environmental thermal stress.

B. INDIVIDUAL ORGAN SYSTEM EFFECTS

The Central Nervous System is the most obvious organ system to see the early effects of hypothermia. Initially, there are subtle decrements in cognition, memory, concentration and dexterity (due to the diminished peripheral nerve function as well as the direct local

effects of cold on muscle function). Thought becomes cloudy in a gradual transition to severe cerebral impairment and unconsciousness as core temperature drops. Reflexes gradually slow and eventually disappear. These include deep tendon and pupillary light reflexes.

The respiratory changes include an initial hyperventilation which, when coupled with the decreased carbon dioxide production from the decreased cellular metabolism, results in low carbon dioxide levels in the blood. Since carbon dioxide blood level is the major determinant of respiratory drive, this lowered carbon dioxide level results in dangerous hypoventilation via suppression of the Central Nervous System respiratory drive. This even further reduces the amount of oxygen supplied to the already oxygen-starved tissues.

Initial cardiac responses are increased blood pressure and an increased heart rate as a result of the vasoconstriction and shivering. As the core temperature drops, the heart rate follows in an almost linear relationship resulting in a declining cardiac output. This further reduces tissue perfusion. However, due to the shunting of blood to the core, the heart and brain retain a greater percentage of their normal perfusion than any other organs. The electrocardiogram shows a classic slowing of all waves and the emergence of an abnormal 'J wave' at around a core temperature of 85°F. At temperatures below 85°F, the heart is prone to ventricular fibrillation which can be initiated by even the slightest trauma. If the heart remains in a regular rhythm, it gradually slows to a stop somewhere below a core temperature of 70°F. This is long preceded by atrial fibrillation. It is important to note the refractory nature of cold-induced arrhythmias to both drug and countershock or difibrillation therapies. Drug therapy is, in fact, contraindicated in the severely hypothermic patient since the slowed hepatic metabolism cannot inactivate these drugs and they would, consequently, accumulate to suddenly have their effect additively and uncontrollably as the patient warmed with therapy.

Gastrointestinal motility is halted. This is postulated to be a contributing factor to the high incidence of pancreatitis after therapy for hypothermia. Gastric stress ulcers are common. The liver, although not very sensitive to permanent injury, rapidly loses its ability to metabolize the body's waste products as well as exogenously introduced drugs. Thus, lactic acid, produced in large quantities by the anaerobically functioning muscles of the extremities, accumulates in large quantities. Large amounts of lactic acid have a detrimental effect on cellular metabolism and will contribute to myocardial irritability.

The kidneys show an initial marked diuresis which contributes to the dehydration and hemoconcentration seen.

The endocrine system is stimulated to maximally increase cellular metabolism.

Hematologic effects include hemoconcentration as a result of a diuresis and of plasma leaking from the vessels into body tissues producing edema. The hemoglobin contained in the red blood cells will less rapidly give up its oxygen to the tissues. Hyperglycemia is initially seen but rapidly converts to a severe hypoglycemia as glycogen stores are utilized for heat production. Electrolyte imbalances are seen, especially hyperkalemia. The blood tends to clot much more readily. This effect, coupled with the slowed blood flow and hemoconcentration, results in small blood clots throughout the body creating stress ulcers in the stomach, strokes in the brain and infarction in the myocardium.

IV. Clinical Presentation

The clinical presentation follows the organ effects detailed above. A correlation with core temperature is intended only as a general guide, noting that effects show considerable individual variation.

Subjectively, the victim will be aware only of the Central Nervous System effects. He will, of course, feel cold but may also be aware of functional deficits in dexterity and sensory perception. Cognitively, thinking will be slowed, memory faulty and logic impaired. His speech will gradually slow. As core temperature drops, so will his level of consciousness until all reflexes have disappeared, voluntary motion ceases and stupor and coma intervene. Occasionally, hallucinations will provide the victim with a mistakenly euphoric sense of well-being.

Objectively, it is very important to accurately ascertain the core temperature. This can be done with either rectal or esophageal determinations since these accurately reflect the core temperature. A special thermometer is available for measuring especially low body temperatures. Of greatest objective concern are the Central Nervous System and cardiac signs. A good mental status exam is essential with the conscious victim. Cardiac signs run the gamut from normal sinus rhythm to ventricular fibrillation and require an electrocardiogram for adequate monitoring in the severely hypothermic patient. Recall that blood pressure and the pulse may be unobtainable (in part due to the peripheral vasoconstriction), reflexes may be absent and pupils fixed and dilated. In short, the patient may meet the ordinary criteria for pronouncement as deceased but he may also benefit from resuscitation efforts. *The CARDINAL RULE is that one is not dead until he is WARM and dead!!*

The corpsman should concentrate his vigilance on early recognition of the subtle signs and symptoms of hypothermia rather than the obvious advanced signs. It will be the mildly hypothermic patient who will present during routine operations. Remember, also, that hypothermia presents a continuum of symptomatology from normal functioning to death with many variations.

V. TREATMENT

The first rule of treatment is to follow standard first aid procedures: the ABC's. Ensure an airway, support adequate breathing and maintain circulation. Next, the secondary first aid measures can be administered to bleeding, fractures, etc.

There are five general rules specific to treatment of hypothermia victims. They are:

1. Rewarm the victim only when it can be done properly.
2. Handle the victim carefully (recall the myocardial irritability which may cause fibrillation with even minor stimulation).
3. Prevent further heat loss.
4. Do not rewarm too quickly.
5. Rewarm the core first.

Before treatment is begun, differentiation must be made between the diagnosis of mild hypothermia and severe hypothermia. Generally speaking, the dividing line should be drawn between the victim who is conscious and shivering and one who is unconscious and/or has ceased shivering. The conscious, shivering victim is considered to be only mildly hypothermic and will not be severely acidotic nor prone to cardiac arrhythmias. Most importantly, he will have retained the ability to rewarm himself if removed from the chilling environment. He can be rewarmed by any of the following methods without risk of serious complications:

a. Removal from the environment.
b. Application of blankets and other warming and insulating materials.
c. Application of a moderate amount of heat to the whole body from a room heater, warm shower or hot packs placed on the 'pressure points'.
d. Ingestion of warm fluids, avoiding caffeine, a vasoconstrictor.
e. Huddling one sparsely clothed person wrapped in a blanket with the victim. This method is of limited value, but better than nothing.

Any of these methods will result in almost immediate improvement in the symptomatology of mild hypothermia. The victim's state of consciousness will improve, shivering will abate and dexterity will return.

In contrast, the severely hypothermic patient will be made precipitously worse by any of the above treatment methods as they would vasodilate the periphery and allow cold, acidotic blood to enter the core before the core could be rewarmed. The result would be a colder, more acidotic victim with greater potential for ventricular fibrillation as this new cold blood insults the myocardium. This will also cause a lowering of the core temperature, an effect called *afterdrop*. The cardinal principle of treatment for severe hypothermia is to rewarm the core first. This is done chiefly by three methods:

Immersion of the trunk only in water at about 105°F. This method allows access to the arms and head for intravenous and inhalation therapies but limits access to the chest for CPR or cardiac monitoring. It is a very effective method.

Inhalation of warmed (110-112°F), moist oxygen delivered by a special portable resuscitator is also very effective and can be used along with the trunk immersion method. A new portable oxygen warming device may soon become available for use in the field. Until that time, however, this method is available only to those in a hospital or research setting.

Peritoneal irrigation with 105°F fluids takes advantage of the large surface area of the abdominal cavity and requires minimal equipment and support. The procedure is performed by a physician and is reserved for use in a hospital setting. As such, it is mentioned here only because it is a very effective method which may be utilized on the corpsman's patient once he reaches the hospital.

Whichever method is utilized, a rewarming rate of about 1°F per hour is about optimum. As a general rule, a victim of hypothermia whose core temperature is below 94°F will require immediate hospitalization and supervision by physicians aware of the special problems involved in treatment. In addition to rewarming the severely hypothermic patient, intravenous therapy is instituted to counter the effects of dehydration, acidosis, electrolyte imbalances, myocardial irritability and cardiac arrhythmias. Ventilatory assistance may also be required. The only method of rewarming the severely hypothermic patient outside of the hospital setting is the trunk immersion method.

VI. Prevention

The key to management of the hypothermia problem is its prevention. There is no magic to it, just common sense. First, keep warm. Second, if you must enter a cold environment, be properly protected. Third, if neither of the first two is possible or they are ineffective, then conserve heat by proven methods.

Keeping warm means allowing prolonged exposure to the cold to only those personnel of whom it is absolutely required. Warm clothing should be worn and should be made of wool and worn in layers to trap more insulating air. Remember to cover the head since vasoconstriction is ineffective in the scalp and up to 80% of the total heat lost can be via the head. Personnel who have been exposed to the cold should rewarm thoroughly upon return to warmer areas. If one must reenter the cold environment soon, it would be better to remain chilled. A hot shower would indeed warm

the periphery, but would also allow cooled blood from the extremities to enter the core and would vasodilate the vessels in the extremities. This vasodilation would allow more rapid cooling once the cold environment was reentered.

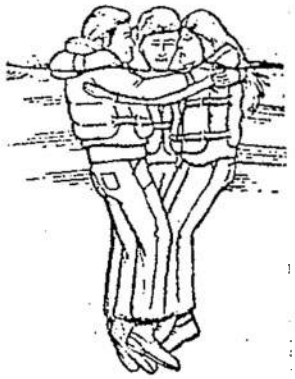
Protection for the diver is essential. Three general types of exposure suits are available; the free-flowing hot water suit, the wet suit and the dry suit. The hot water suit operates by having heated water pumped down from the surface to flow through the diver's suit. The wet suit relies on air trapped in the closed-cell neoprene for insulation. This type of suit is, however, compressed as the diver descends and is of little insulating value at significant depths. The dry suit (the Unisuit and the MK V Deep Sea Diving Dress) is kept inflated by the diver as he adjusts the supply of his breathing air to it. Its insulating value is derived from the diving underwear worn beneath the suit and, consequently, is of limited value in really cold water.

Adequate surface intervals for the diver are essential. Thorough rewarming is the rule and a guideline is that one is adequately rewarmed when sweating begins.

Finally, if all else fails, or the environmental effects of the cold surpass the body's efforts to maintain core temperature, there are additional methods of heat conservation more accurately termed survival than preventive methods. It is essential that the head be kept out of the water. Drownproofing is a proven method for sustaining oneself on the surface for long periods of time. However, it is accomplished by remaining vertical and keeping the head fully immersed in the water. Drownproofing is, therefore, a dangerous practice since is would allow rapid heat loss from the immersed head. Instead, the H.E.L.P. (Heat Escape Lessening Position) should be employed. H.E.L.P. is the position in which the least amount of body surface area is presented to the chilling water. The legs are crossed and tucked toward the chin with the arms wrapped around the legs or a flotation device. If possible, the hands should be placed in the axillae. If there is more than one in the water, the Huddle position is best. This is accomplished by remaining vertical in the water, each person holding the others tightly with his arms. Survival time in the water is dependent largely on two factors; water temperature and the behavior of the victim!! Flotation gear should be worn at all times when working near the water!

Remembering these common sense preventive measures will not only decrease the risk but will also increase the enjoyment of working in a cold environment.

The H.E.L.P. Position



The Huddle Position

SIGNS OF HYPOTHERMIA AS APPROXIMATELY RELATED TO CORE TEMPERATURE

°C	°F	
37.6	99.6	'Normal' Rectal Temperature
37	98.6	'Normal' Oral Temperature
36	96.8	Metabolic Rate Increases - trying to overcome heat losses
35	95.0	Shivering Maximum
34	93.2	Victim still conscious with normal blood pressure
33	91.4	*SEVERE HYPOTHERMIA BELOW THIS LEVEL*
32	89.6	Consciousness becomes clouded, pupils dilate but remain reactive to light, blood pressure becomes difficult to obtain.
31	87.8	
30	86.0	Progressive loss of consciousness, muscular rigidity increased, pulse and blood pressure very difficult to obtain.
29	85.2	
28	82.4	Ventricular fibrillation may develop if heart irritated, cardiac arrhythmias develop, atrial fibrillation occurs spontaneously.
27	80.6	
26	78.8	Victim seldom conscious
25	77.0	Ventricular fibrillation may occur spontaneously
24	75.2	Pulmonary edema develops
23	73.4	
22	71.6	Maximum risk of ventricular fibrillation
21	69.8	
20	68.0	Cardiac Standstill
19	66.2	
18	64.4	
17	62.6	Flat-line Electroencephalogram (EEG)
16	60.8	
15	59.0	Lowest accidental hypothermia victim to recover
9	48.2	Lowest artificially cooled hypothermia patient to recover

SUMMARY OF HYPOTHERMIA TREATMENT MODALITIES AVAILABLE TO THE CORPSMAN IN THE FIELD

MILD HYPOTHERMIA:

1. Remove the victim from the chilling environment.
2. Apply blankets and other external warming and insulating materials.
3. Apply external heat in the form of warm showers, radiant heat, hot packs to 'pressure points'.
4. Ingestion of warm fluids, avoiding caffeine.
5. Huddle if no other means is available.

SEVERE HYPOTHERMIA:

	Advantages	Contraindications	Disadvantages
1. Trunk Immersion	Readily available Excellent result Allows preferential core rewarming	Trauma with open wounds CPR necessary	Chest inaccessible for CPR Difficult to monitor heart and lungs Must have large basin or tub
2. Inhalation	Leaves chest available for CPR Very effective Selective rewarming of heart and lungs Curtails respiratory heat loss Can be combined with #1 Simple equipment which is inexpensive, easy to transport and requires no external power	Severe trauma to the face (although tracheotomy or intubation can be performed)	Patient must have spontaneous respiration (although tracheotomy or intubation can be performed)

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hypothermia; cold weather; immersion; heat loss

20. ABSTRACT *(Continue on reverse side if necessary and identify by block number)* Hypothermia connotes the abnormal lowering of body core temperature (the internal temperature of the body; that of the core organs) the effects of which are in a gradient from only a mild decrease in motor and cognitive functions to the severe reactions of cardiac and respiratory failure. Hypothermia becomes important to the corpsman who will accompany personnel into the thermally-stressful environments of cold weather and cold water.

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Item 20--continued

This presentation is designed to provide the corpsman with a basic understanding of the body's normal thermoregulatory mechanisms, their methods of adaptation to cold stress and the clinical syndrome which results. Knowledge of the basic pathophysiology will allow the corpsman to interpret the subtle objective indicators of hypothermia and to recognize the vague subjective indicators as voiced by the victim. This background will facilitate prompt treatment and relief of a potentially dangerous malady.