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FINAL REPORT

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16. Abstract

This report summarizes medical and scientific aspects of the general literature debate over rapid versus slow rewarming and selected specific rewarming approaches. The specific approaches were selected largely on the basis of being difficult to evaluate with in vivo experiments with human subjects. Findings in basic medical literature, research reports and clinical medical literature are analyzed leading to recommendations of the suitability of each specific therapy for use as a first-aid treatment in the field.

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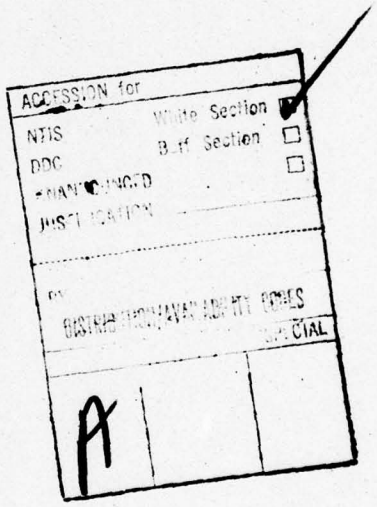
PREFACE

This final report documents the work conducted under Task Number 5 of Contract Number DOT-CG-72074-A from January 17 to October 30, 1978. The work was performed at Clemson University under the auspices of the U.S. Coast Guard, with LTjg Steven F. Wiker serving as program monitor. The principal investigators were Drs. R. Michael Harnett, Fred R. Sias, and James R. Pruitt.



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A LITERATURE REVIEW

for

UNITED STATES COAST GUARD
U.S. COAST GUARD HEADQUARTERS

Contract No. DOT-CG-72074-A
Task Number 5

from

Clemson University

by

R.M. Harnett, Ph.D., F.R. Sias, Ph.D., J.R. Pruitt, M.D.

February 14, 1979

1.0 INTRODUCTION

The literature on hypothermia resuscitation is voluminous. It includes a large number of clinical observations as well as basic physiological and medical experiments. As with much of clinical medicine, there are controversies concerning the proper choice of therapy for resuscitating patients from the hypothermic state.

Hypothermia could be defined as "the clinical state of subnormal body temperature..." (Benazon, 1974). However, "normal temperature" and "body temperature" are not precise terms since normal temperature has a diurnal variation of 1 to 2 degrees centigrade. In addition, measurements taken at different body locations show differences at any given time. Skin temperature (even in protected locations such as the armpit) is almost always different from deeper body locations such as rectal temperature, esophageal temperature, tympanic temperature, or the temperature control location in the preoptic area of the hypothalamus. Much of the experimental and clinical literature is based on rectal temperature measurement which is affected by probe depth and possibly external factors such as blood flow returning from the legs. Despite the inadequacies of rectal temperature, it has been used as a primary indicator of deep-core temperature of the body.

Most clinical and research investigators would probably agree that a rectal temperature below 35°C is indicative of "clinical hypothermia" and that a temperature below 32°C is indicative of "severe hypothermia".

The lowest recorded rectal temperature with survival from accidental hypothermia is 18°C. Patients with a rectal temperature above 32°C will normally recover spontaneously if removed from the cold environment and there are not other underlying factors causing hypothermia (Weyman, et al., 1974). Most resuscitation measures should be aimed at reviving hypothermia victims with rectal temperatures below 32°C.

Table 1 illustrates the levels of hypothermia and their associated physiological characteristics. The effectiveness of resuscitative measures will be closely coupled to physiological states at different rectal temperatures. It should be noted that there is some variation in the temperatures at which the literature indicates major changes to occur. Table 1 represents an attempt to distill a consensus from this variation.

Death due to hypothermia is generally attributed to ventricular fibrillation in man (Alexander, 1945; Hervey, 1973; Swan, et al., 1955) although this is not a proven fact since direct evidence, such as an EKG recording, is seldom available at the time of death. The Dachau experiments (Alexander, 1945; Gagge, and Herrington, 1947) and some animal experiments (Bigelow, et al., 1950a) strongly suggest that ventricular fibrillation is the terminal event if patients are not maintained by cardiac massage and artificial respiration. Some animal experiments (Popovic and Popovic, 1974; Hamilton, et al., 1937) point to respiratory failure as a terminal event but this is not considered to be the case in man. Resuscitation of a hypothermia victim involves a number of supportive and restorative measures some of which are conflicting. Table 2 lists the principal changes that must be affected in restoring a person to normothermia. Each rewarming therapy considered in this report will be examined from each of these viewpoints.

There is an extensive body of evidence indicating that seriously cooled hypothermia victims should be handled carefully (Burton and Edholm, 1955; Truscott, et al., 1973; Bigelow, 1959). By "carefully" it is meant that the muscular exertion of the victim should be minimized. Even the process of climbing aboard a ship may release pooled cold blood causing a continuing fall in core temperature (afterdrop) and possibly fatal ventricular fibrillation. Afterdrop is generally thought to be possible only when there is a source of

TABLE 1
LEVELS OF HYPOTHERMIA

°F	°C	
99.6	37.6	"Normal" Rectal Temperature
98.6	37	"Normal" Oral Temperature
96.8	36	Increased Metabolic Rate in attempt to balance heat loss
95.0	35	Shivering maximum at this temperature
93.2	34	Patients usually responsive and normal blood pressure
91.4	33	SEVERE HYPOTHERMIA BELOW THIS TEMPERATURE
89.6	32	Consciousness clouded — Pupils dilated
87.8	31	Blood Pressure difficult to obtain — most shivering ceases
86	30	Progressive loss of consciousness; Increased muscular rigidity
85.2	29	Slow pulse and respiration
82.4	28	Cardiac arrhythmia develops; Ventricular *fibrillation* may develop if heart irritated
80.6	27	Voluntary motion lost along with pupillary light reflex, deep tendon and skin reflexes - appear dead
78.8	26	Victims seldom conscious
77.0	25	Ventricular fibrillation may appear spontaneously
75.2	24	Pulmonary Edema develops
73.4	23	
71.6	22	Maximum risk of fibrillation
69.8	21	
68.0	20	Heart Standstill
66.2	19	
64.4	18	Lowest Accidental Hypothermic patient with recovery
62.6	17	ISO-ELECTRIC EEG
48.2	9	Lowest Artificially Cooled Hypothermic patient with recovery

TABLE 2

REQUIREMENTS FOR RESUSCITATING HYPOTHERMIA VICTIMS

1. Maintain adequate cellular respiration
   (a) maintain adequate circulation
   (b) maintain suitable ventilation
2. Minimize additional heat loss
   (a) remove patient from cold
   (b) prevent other routes of heat loss
3. Restore normal body temperature
   (a) apply heat
   (b) stimulate normal body sources of heat
   (c) restore all lost calories
4. Restore normal body homeostasis
   (a) establish normal pH
   (b) establish normal blood glucose levels
   (c) restore proper kidney function
   (d) restore normal fluid and electrolyte balance
5. Treat any non-environmental cause of hypothermia
6. Treat medical problems unrelated to hypothermia

blood significantly colder than the body core; that is, when a significant temperature gradient exists between body core and periphery.

There is also evidence from anecdotal clinical experience and surgical hypothermia which suggests that the only measure of death is whether or not a person can be resuscitated, regardless of the body temperature (Golden, 1973; Jessen and Hagelsten, 1972; Stewart, 1972, Nemiroff, et al., 1977).

During the three decades since World War II a number of rewarming therapies have been proposed for resuscitation of hypothermia victims. These range from simple hot showers, heating pads and trunk immersion in hot water to the inhalation of hot moist gasses, diathermy, peritoneal irrigation, intragastric balloons, extracorporeal blood warming and plumbed garments circulating warm fluids.

In addition to these specific rewarming therapies two general approaches to rewarming have attracted continuing debate. One pursues the rapid restoration of lost heat through the application of active measures. The other seeks to slowly restore body heat through the use of passive measures.

This report presents a picture of the rewarming problem and then summarizes the open literature debate over rapid versus slow rewarming. The significant literature information concerning selected specific rewarming therapies is presented.

The specific therapies addressed were selected because they are particularly difficult to evaluate by direct human research or because their application in profound hypothermia poses particular problems. The therapies are described in some detail, their advantages and risks are discussed qualitatively, known contraindications for the therapies are delineated and discussed, and specific guidelines for their application are summarized. Finally, conclusions are drawn concerning the use of the therapies as first-aid treatments by emergency medical technicians (EMT's). These conclusions are based strictly on scientific and medical considerations. No legal implications of these conclusions were considered. In addition to previously published facts and opinions, medical and scientific opinions of the authors have been included in this report.

2.0 THE REWARMING PROBLEM

This chapter presents the problems faced in rewarming victims of profound hypothermia. The problem is first characterized in general terms and then in detail by summarizing scientific and clinical observations relating to physiologic anomalies which may affect the choice of a rewarming therapy.

2.1 General Nature of the Problem

Based on the current literature, the total rewarming problem may be stated simply as the restoration of all lost heat without precipitating additional fatal side effects.

There is a growing body of evidence that hypothermia per se may not be fatal above a rectal temperature of around 25°C. Below about 25°C ventricular fibrillation may appear spontaneously. There is evidence in dogs (Hughes, 1956) that even ventricular fibrillation may not be fatal for periods of over one hour if appropriate rewarming and defibrillation is carried out. During cardiac surgery man has survived circulatory standstill for long periods of time. Nemiroff, et al., (1977) described 11 cases of cold-water near-drowning. They indicate that physiologic responses to cold-water immersion serve to prolong life, even during a 38-minute cessation of respiration. Their work is summarized in the August, 1977, Scientific American (page 57). Niazi and Lewis (1958) report recovery of a woman from 9°C with no cardiac activity or respiration for one hour.

If some circulation is maintained by closed chest massage and the lungs are ventilated, patients can survive for extended periods at very low temperatures. It has been concluded (Jessen and Hagelsten, 1972) that the only sure indication of death from hypothermia is the inability to resuscitate the patient. Nemiroff, et al., (1977) recommends continuing resuscitation efforts, "until the patient is rewarmed and still does not respond."

If a patient is unconscious with shivering suppressed or absent, it may be assumed that the patient is severely hypothermic and requires definitive treatment. A moderately hypothermic patient still shivering but with clouded consciousness may become severely hypothermic if left untreated or mishandled.

This report examines the effectiveness of methods for treating profound hypothermia as generally characterized by rectal temperatures below

32°C, depressed rates of respiration, acidosis, hypovolemia (reduced blood volume), clouded consciousness or unconsciousness, generally cooled organ systems and irritable myocardium. It will be assumed that a significant temperature gradient exists in the victim's body between core and periphery and that he is therefore susceptible to afterdrop. The following paragraphs outline specific aspects of the rewarming problems with which a therapy must contend.

2.2 Fluid Shifts

In cold exposure, diuresis (increase in urine output) occurs in spite of diminished glomerular filtration and renal blood flow (Tansey, 1973). This serves to diminish blood volume; but Hervey (1973) cites Moyer, et al., (1956) and Kanter (1962) who observe that changes in renal function due to cold exposure tend to be masked by circulatory changes. According to Keatinge (1969) cold diuresis, "is probably a physiological mechanism for removing some of the blood volume when this becomes excessive as a result of constriction of blood vessels in the cold." Hervey (1973) called it, "a volume regulatory response to increased filling of deep capacitance vessels."

There is considerable evidence that blood volume decreases as patients and animals are cooled to low temperatures. According to Popovic and Popovic (1974) the blood volume decreases, "to 60 percent of normal volume after 6 to 8 hours at a body temperature of 15°C." They also indicate that, "the circulating blood volume increases during rewarming." The increase in volume may be up to 130 percent of the value prior to cooling.

A number of authors report that the decrease in blood volume is accompanied by an associated concentration of the blood (Popovic and Popovic, 1974; Hervey, 1973; Burton and Edholm, 1955; Keatinge, 1969). The hemoconcentration results in hematocrit approaching 60 percent (Hervey, 1973) and blood viscosity increasing 2 percent for each 1°C decrease in temperature (Hedley-Whyte, et al., 1976). Barbour, et al., (1944) attribute hemoconcentration to shifts of fluid into cells. They suggest that increased cellular activity in the cold results, in the production of osmotically active metabolic end products that require the movement of water into the cells. However, Popovic and Popovic (1974) conclude, "the mechanism of hemoconcentration induced during cooling is not well understood."

There is also evidence (Barbour, et al., 1944) that fluid shifts into cells are reversed producing increased extracellular fluid when the central nervous system becomes sufficiently chilled to abolish reflex responses to cold.

Tansey (1973) cautions that, "While one expects a hemoconcentration both from contraction of plasma volume and from fluid shifts, hemodilution can occur with blood loss, with parenteral fluids, or with aspiration of hypotonic water."

There are indications that the severity of fluid shifts is directly related to the duration of cold exposure and, therefore, to the rate of onset of hypothermia. Tansey (1973) cites slow rewarming as appropriate for "prolonged cold exposure (greated than 6 hours)", in part, because of "hemoconcentration from fluid shifts". Keatinge (1969) indicates that excretion of water can produce a, "serious loss of blood volume during prolonged hypothermia." Burton and Edholm (1955) contrast acute and chronic hypothermia as follows: "In chronic hypothermia...the duration of exposure produces marked changes in blood volume and depletion of glycogen reserves." Any problems which arise from the shift of fluids, such as the tendency toward hypovolemia and hemoconcentration, may be expected to be worse if a given level of hypothermia was arrived at through slow onset than if through rapid onset.

2.3 Edema

Edema has been considered one of the complications of hypothermia and may be a significant factor in the choice of a rewarming therapy. Edema is a frequent finding in clinical medicine. Pulmonary edema, cerebral edema and peripheral edema, are three separate entities with different etiologies. Pulmonary edema is generally associated with either pulmonary capillary membrane damage, left-heart failure, or mitral valvular disease (Guyton, 1976; Beeson and McDermott, 1971) but also accompanies anoxia. Peripheral edema may have several causes including a decrease in plasma proteins, increased capillary permeability, right-heat failure, and "pore stretch" due to increased capillary pressure. Guyton (1976) points out that edema resulting from right-heart failure generally requires fluid retention by the kidneys. Cerebral edema commonly results from a direct injury to the brain.

Critchley (1943), in his study of Shipwreck Survivors, describes sailors having to cut boots off of swollen feet. However, "immersion foot" may be directly responsible for this edema rather than whole body hypothermia. Immersion foot results from, "prolonged soaking of limbs in water at 15°C or below" (Critchley, 1943).

The etiologies of the types of edema that are reported in hypothermia are not clear. It is also not clear whether edema, particularly pulmonary edema, is a consistent finding in acute immersion hypothermia, although pulmonary edema is a frequent complication in elderly individuals found in the hypothermic state.

Pulmonary Congestion and Edema

Barbour, et al., (1944) report earlier work (Walther, 1862) describing edema in the lungs of rabbits dying from hypothermia. They also cite Talbott (1941), who described a similar congested condition of the lungs in the body of a patient who died of hypothermia.

Woodruff (1941) describes experiments in hypothermia using dogs. He reports that one dog out of eleven showed acute pulmonary edema while the hearts of eight showed edema.

The Dachau experiments (Alexander, 1945) report:

> "a great number of experimental subjects showed a profuse oversecretion of mucus, with vesicular foam at the mouth reminiscent of that seen in pulmonary edema. However, there were not other definite clinical signs of pulmonary edema, and auscultation showed merely sharpening and impurity of breath sounds. This foaming at the mouth sometimes appeared as an early symptom at 32°C - 35°C of body temperature. It had no prognostic significance with regard to the fatal or non-fatal outcome of any one experiment...."

A monograph by Lathrop (1972) describes the terminal symptoms of a hypothermia victim on Mt. Adams in 1966: "A terminal event was labored breathing which produced a frothing foam at the mouth. This, we now realize was characteristic of the terminal pulmonary edema of acute hypothermia." Lathrop cites, "probable edema and hemorrhage in lungs" as associated with rectal temperatures below 78°F (25.6°C). He states that "The frequency with which pulmonary edema is present in fatal cases of acute hypothermia, is a new finding in the medical literature on this subject. Often one of the terminal events before death is labored breathing in which a whitish froth may well into the mouth from the congested lungs." He further states, without reference, that an Oregon pathologist, Dr. Warren Hunter, "found evidence of pulmonary hemorrhage and edema in each of 10 fatal cases of accidental hypothermia which he reviewed." He is also said to have, "found that pulmonary edema is very often present as a terminal symptom in such deaths."

Tansey (1973), in prescribing treatment for "whole body hypothermia", says that, "In view of depressed respiration and probably atelectasis with aspiration or anoxic pulmonary edema, oxygen supplement and cautious pulmonary toilet with

endotracheal suction and intermittent positive pressure breathing should be initiated."

There is another possible explanation for the pulmonary congestion described by Talbott (1941), Alexander (1945) and Lathrop (1972). The ciliated cells which line the nasopharynx and the tracheobronchial tree, save for the terminal bronchioles and alveoli, serve to clean these respiratory passages by propelling mucus and waste material toward the mouth. They are normally very effective. Bouhuys (1977) indicates that, "In airways with mucus-secretory elements, the cilia move in thin fluid and are covered with a mucous layer. This two-phase liquid system ... is essential for mucociliary transport." Best and Taylor (1961) indicate that,

> "The efficiency of the cilia depends in part on the viscosity and stickiness of the material which is in contact with them. Their effectiveness may be varied by changing the properties of this material as well as by an increase or decrease in the rate or force of their beating."

Cherniack, et al., (1972), indicate that, "Ciliary activity carries particles and macrophages ... to the larger bronchi where the cough reflex is important in their clearance." They further indicate that the cilia beat, "at a rate of approximately 1000 to 1500 times each minute..." and that, "this causes the mucus to flow toward the glottis at the rate of approximately 10 to 20 mm/min." According to Best and Taylor (1961), "The cilia are not influenced by nerve impulses, but are very susceptible to chemical changes in the blood.... Ciliary action is depressed by cold and increased when the temperature of the cells is raised slightly above normal." Bouhuys (1977) cites Iravani (1967) indicating: "In a 0.2-mm diameter rat airway, beat frequency was 13/sec at 35°C, and 2/sec at 20°C..." and Hakansson and Toremaln (1965) indicating: "in the rabbit treachea, 20/sec at 37°C, and 7/sec at 20°C." Boujuys also indicates that, "mucus has plastic and elastic, as well as viscous, properties." He states that, "In conformance with a property of plastic materials, a certain minimum stress must be exerted before mucus moves at all." This supports the notion that a thermally-induced slowing of ciliary activity could not only slow mucociliary transport, but could actually stop it completely. Clearly, the depressing effects of hypothermia upon cilia activity can allow the accumulation of mucus secretions in the respiratory tree presenting a clinical picture resembling pulmonary edema. Of course, the possibility should not be overlooked of the simultaneous existance of pulmonary edema and mucus accumulation due to cold-induced cilia inactivity.

Cerebral Edema

The literature on cerebral edema is also conflicting. According to Popovic and Popovic (1974), "There are several reports that hypothermia protects against cerebral edema..," (Clausen, et al., 1970; Fay, 1959). The Popovics also cite Herrmann and Dittmann (1970) who mention indications of cold-induced swelling of the brain.

According to Khalil (1957), cerebral edema, "has been reported to follow the use of [extracorporeal blood cooling]."

The Dachau report (Alexander, 1945) also shows that autopsies revealed, "edema of the brain and marked congestion of all cerebral vessels" in "those cases where there had been additional cooling of the neck and occiput." The Dachau experiments also point to an increase in spinal fluid pressure in cases with marked additional cooling of the neck and occiput.

Hedley-Whyte, et al., (1976), cite Bloch (1967b) cautioning that,

> "Relapse into coma or confusion can occur with rewarming, but improvement occurs if the temperature is allowed to fall again. These changes appear to be the result of an increase in cerebrospinal fluid pressure. Patients who become increasingly drowsy during rewarming promptly become more alert when cerebrospinal fluid pressure is reduced by lumbar puncture with removal of cerebrospinal fluid. Some patients who develop acute pulmonary edema during rewarming experienced dramatic relief within a few minutes of reduction of cerebrospinal fluid pressure."

Peripheral Edema

Barbour, et al., (1944) concluded from experiments conducted with twelve monkeys (Macacca Mulatta) cooled to 23°C and rewarmed, "If any edema is present in monkeys at these low body temepratures, we were unable to establish it definitely, although some apparent swelling was noted about the shoulders and forearms in one case." Their results with rats were quite different. They report: "At 16°C, the rats were completely prostrated in a condition resembling deep anesthesia. At this point considerable edema of the face, eyelids, and jaw as described by Hamilton [1937], was a constant finding." They conclude: "Subcutaneous edema tends to occur and is augmented on rewarming the animal, which procedure increases greatly the hydration of blood, at least relatively."

A paper on accidental hypothermia (Duguid, et al., 1961) review twenty-three cases of hypothermia. Most were elderly individuals with associated,

"states of disability and illness" including respiratory infection and heart failure. Duguid states: "Peripheral edema was present in over half the cases."

Summary on Edema

Edema per se is not mentioned in Burton and Edholm's monograph "Man in a Cold Environment' (1955) in regard to immersion hypothermia but they do mention a, "fluid shift from the plasma to the tissue." Keatinge (1969) acknowledges the possible contribution of "increased vascular permeability" to a, "loss of blood volume during prolonged hypothermia." As indicated in Section 2.2, Barbour, et al., (1944) attributed hemoconcentration to shifts of fluid into cells which may reverse themselves later in cooling producing increased extracellular fluid.

It is reasonable to speculate that edema observed in conjunction with hypothermia might be connected etiologically with the other fluid shifts discussed in Section 2.2. If this connection exists, one may anticipate that the severity of edema would be worse for hypothermia of long duration than short duration.

Edema, either pulmonary, cerebral, or peripheral is not consistently reported in the literature on immersion hypothermia. Yet there are sufficient references to observations of edema and to related mechanisms to preclude its dismissal as a physiological anomaly unrelated to cold exposure. An implication of this conclusion is that rewarming therapies intended for treatment of profound hypothermia should be analyzed for their effects on edema.

2.4 Electrolyte and Acid-Base Changes

Electrolyte Changes

There have been numerous studies of electrolyte changes during experimental hypothermia but the results are conflicting and controversial. Serum potassium has been found to increase in dogs (Smith, 1956; Bigelow, et al., 1950b) and in man (Langdon and Kingsley, 1964), to remain the same in dogs (Axelrod and Bass, 1956), and to decrease in dogs (Kanter, 1963; Gollan, et al., 1957) and in rabbits (Grote and Schweikhordt, 1969). If the potassium

concentration could be shown to rise consistently, this might explain the increased cardiac irritability and the tendency toward ventricular fibrillation that is often found in hypothermia victims. Popovic and Popovic (1974) further point out, "The potassium ion concentration in hypothermia can be compensated by glucose administration which tends to return the excitability of the hypothermic heart to normal levels." The reference cited for this is Spurr, et al., (1959). Jessen and Hagelsten (1978) report that, "plasma electrolytes are usually normal when the treatment starts, but during the last phase of the rewarming, a severe hypopotassemia is often provoked." They conclude that, "it may well be responsible for some of the deaths which are reported after the active rewarming has stopped and the body core temperature has reached almost normal values."

Alterations in potassium distribution are generally assumed to be due to changes in membrane permeability and/or alterations in the sodium-potassium pump due to hypothermia (Popovic and Popovic, 1974). They further state, "In moderate hypothermia there is no change in serum sodium concentration, but the plasma level falls when the body temperature of an experimental animal is decreased below 25°C."

Hyperphosphatemia induced in rats by hypothermia has been reported (Popovic and Popovic, 1974). Calcium changes are as varied in the literature as potassium with serum levels found to be elevated in dogs (Axelrod and Bass, 1956; Smith, 1956), unchanged in rats (Nowell and White, 1963), and decreased in rabbits (Grote and Schweikhordt, 1969). The magnesium concentration in the plasma was reported to rise in rats during hypotheria (Nowell and White, 1963).

In summary, Popovic and Popovic (1974) state, "Hypothermia induces some changes in the electrolyte metabolism, but the overall picture is still quite obscure.... Different techniques of cooling, different species of animals, and the precooling state of experimental animals or patients greatly affect the results obtained during cooling.

Tansey (1973) cautions that, "Blood electrolytes must also be interpreted with cognizance of ...fluid shifts [which have occurred]."

Adic-Base Changes

The literature on acid-base balance has also been summarized by Popovic and Popovic (1974),

> "During cooling and rewarming the pH value of the blood of experimental animals or of patients decreases (cold acidosis), sometimes

even when bicarbonate is adminstered. Both respiratory and metabolic acidosis develop during cooling. This acidosis is very pronounced but does not appear to be harmful. However, during surface cooling, a much decreased pH of the blood might be associated with the increased tendency toward ventricular fibrillation."

They also state, "During rewarming, metabolic acidosis occurs even if it were not observed during the cooling process. However, the acidosis disappears after rewarming is completed."

Keatinge (1969) cites as causes of acidosis:

1. failure of acid excretion,
2. respiratory depression,
3. increased solubility of CO_2, and
4. lactic acid forming faster than it can be metabolized during shivering.

He concludes that, "All these factors together do not normally cause dangerous acidosis while the body temperature remains low"; but he cites Fairley, et al., (1957) indicating that "severe acidosis may develop during rewarming."

3.0 RAPID VERSUS SLOW REWARMING

The two general approaches to rewarming loosely referred to as rapid and slow rewarming have been often debated. Each therapy has its proponents and its detractors. W.R. Keatinge and A.R. Behnke support rapid rewarming while H. Hillman advocates slow rewarming. This chapter presents a cross section of literature representing the continuing debate over the better approach to rewarming.

3.1 General Discussion

The effort to avoid side effects during rewarming has resulted in two general schools of thought, each with sub-categories. The two dominant approaches to hypothermia resuscitation may be termed "rapid rewarming" and "slow rewarming". There appears to be general agreement that intermediate warming rates should be avoided (Burton and Edholm, 1955, Hervey, 1973).

The extremes of viewpoints are illustrated by Bloch (1967a) who states "There does not appear to be any harm resulting from slow rewarming following prolonged hypothermia" and Meriwether and Goodman (1972) who state, "Rapid rewarming by water immersion is recommended for treating patients with severe accidental hypothermia." Fernandez, et al., (1970) point out that the main controversy is not between core rewarming and external rewarming but between "passive external rewarming" and "active external rewarming".

The review by Fernandez, et al., (1970) of the literature regarding fast and slow rewarming is particularly appropriate:

> "The major controversy has been between the use of passive external rewarming and active external rewarming. Active external rewarming means the direct application of exogenous heat directly to the surface of the body while passive rewarming utilizes the heat produced by the patient without the addition of artificial heat to the body surface. The authors with the greatest experience have favored slow, usually passive, external rewarming.
>
> "In 1958, Emslie-Smith, described eight patients with accidental hypothermia, all of whom died. In only three cases was the method of rewarming recorded. Those three patients were rewarmed by electric blanket. It is of note that rewarming was slow. No patient was rewarmed in less than 12 hours.

"Duguid and co-workers, in 1961, reported 23 cases of accidental hypothermia. Six patients were 'actively' rewarmed and all died. Seventeen patients were passively rewarmed. Ten patients died during rewarming and an additional four died later in the hospitalization, yielding a total mortality of 87%. Again, the important point is that none of the 23 patients were rewarmed in less than 24 hours. The authors concluded from these results that passive rewarming was the method of choice in their hypothermic patients.

"In 1962 Prescott and his colleagues, rewarmed nine patients passively over prolonged periods of time and six died.

"Fruehan [1960] treated eight accidentally cooled patients. Four were rewarmed actively and four passively. One of the eight survived. Although the survivor was passively rewarmed, the author emphasizes that this patient had the most rapid rate of rewarming.

"Recently, Maclean and associates [1968] reported enzyme changes in 25 accidentally frozen patients. Ten patients recovered. While the method of rewarming was not stated, all four patients who were rewarmed in less than 12 hours survived.

"The results of human experiments performed on persons of block 5 of Dachau in 1942 support the rapid active rewarming method. These sordid investigations proved to the satisfaction of the executioners that the best method of resuscitating hypothermic prisoners was by rapid and intensive rewarming."

A variation on the simplified rapid vs. slow debate are the following steps recommended by Bangs (1976) for treatment of chronic hypothermia (partial list):

1. Rewarm the victim only when it can be done properly.
2. Handle the victim gently.
3. Prevent further heat losses.
4. In the field <u>do not</u> rewarm too fast.
5. Rewarm the core first.

He recommends rewarming in a hospital, "If at all possible", or a, "safe environment where ...[the victim] can be rewarmed properly with proper monitoring, equipment and medicine...." Further he indicates that if rewarming has to be done in the field, "it is probably safest to do it slowly. Slowly

means most anything except submerging the victim in hot water." His objective is to avoid, "blood vessel dilation and shock...."

3.2 Spontaneous or Passive Rewarming

Slow spontaneous (or passive) rewarming has long been advocated as the proper therapy for hypothermia at least in chronic hypothermia of a slow onset and long duration (Rees, 1958; Whitby, 1964; Maclean, et al., 1974). Talbott and Burton (1952) recommend slow rewarming at a rate of about 1/2°C per hour. Bloch (1967a) recommends even slower rewarming such as 1°C in 400 minutes. Bloch also recommends that too rapid spontaneous rewarming be slowed by drugs such as chlorpromazine.

The rationale behind such slow rewarming protocols is that it will avoid rewarming hypotension and afterdrop of deep body temperature perhaps due to cutaneous vasodilatation, and the return of cold peripheral blood to the body core. In addition, slow rewarming is said to allow fluid shifts due to hypothermia to be corrected.

Maclean, et al., (1974), referring to Talbott and Burton (1952), points out that the body tries to restore normal temperature at all levels of hypothermia above 23.8°C. Part of the corrective mechanism is shivering which may be present intermittently down to a temperature of 23.8°C.

Much of the literature supporting slow rewarming involves clinical cases where patients are either elderly and/or have consumed considerable alcohol. The survival rates in these populations are not good; but it is not reasonable to ascribe them to rewarming rate.

Spontaneous rewarming is certainly a viable therapy in mild hypothermia. Truscott, et al., (1973) state, "Bringing the patient into a warm environment and conserving body heat (passive rewarming) will usually suffice." He concludes, "The optimal method of rewarming is still controversial.... Most authors, however, believe that passive surface rewarming is less hazardous."

Tansey (1973) concludes that, "Slow rewarming becomes appropriate with prolonged cold exposure (greater than 6 hours) where hemoconcentration from fluid shifts, increased blood viscosity, exhaustion of glycogen stores, and acidosis all contribute to an upset 'milieu interieur' which will be aggravated by rapid rewarming and which will revive spontaneously if further cold is prevented."

Passive slow rewarming is not as simple as it might seem at first glance. In the hospital environment, a technique generally used is to place the person in a bed, lightly covered, at normal room temperature. Normal room temperature, of course, varies with British and European rooms being often maintained at lower temperatures than those in the United States. If the room temperature is close to the lowered body temperature of the patient and the air is still, little heat will be lost by convection, conduction, or radiation. Extremities may be below room temperature making possible a net gain in heat from the environment.

Some heat will be lost from the body through respiration due to the heat that must be expended to warm and humidify the room air. Heat loss by respiration will be moderate if the room humidity is high and room temperature is not much below body core temperature.

The heat that is to be used in passive rewarming is largely from metabolic activity, which may be less than 50 percent of normal at body temperatures below 25°C, with small amounts being contributed by the environment. At temperatures below about 30°C shivering will be largely suppressed and the sole source of heat is the depressed basal metabolism of the patient. At temperatures above about 32°C shivering will become the dominant source of heat for a spontaneously rewarming patient. The heat generated by shiverng may be several times greater than the resting metabolic rate if the patient did not exhaust his muscle energy supplies during cooling. If energy supplies were severely taxed during the patient's cooling period, shivering may not be able to contribute significantly to a spontaneously rewarming patient.

If spontaneous rewarming is being used as a first-aid measure, the situation may be quite different than described above. A blanket or a sleeping bag intended to prevent further heat loss will temporarily draw heat from the patient if it has not been pre-warmed. Generally, insulation does not conserve heat until the surface layer of the insulation has been raised to body temperature.

A second significant factor in a rescue scenario is that air temperature may be very cold. The heat lost through respiration may be a large part of the total heat production of the body under conditions of no shivering, a basal metabolic rate depressed below 50 percent, and continuing significant heat loss by warming and and humidifying cold, dry ambient air. Spontaneous rewarming would be most unsatisfactory under these conditions. If the patient is shivering, spontaneous rewarming may be completely adequate.

Another consideration is contrary to intuition. Mild surface rewarming methods such as hot water bottles, heating pads, and even body to body heat exchange in a sleeping bag, can suppress shivering, by warming the skin, and thereby remove a rewarming mechanism more effective than the mild surface heat. Of course, it is not possible to make a conclusive statement since such relatively mild surface heat might not suppress shivering in the middle temperature ranges of hypothermia. At lower temperatures, say below 28°C, shivering is already suppressed and mild surface heat will have little effect on the rate of spontaneous thermogenesis. At temperatures above about 30°C suppressing shivering may slow rewarming, but the patient will probably be in no danger anyway and the surface heat will make him feel better. Of course, in all of this discussion it has been assumed that major routes of heat loss are removed.

The major unanswered question is whether a rapidly cooled, deeply hypothermic patient with suppressed shivering can be helped by relatively mild application of surface heat as a first-aid procedure. Since human studies can not be extended to temperatures where shivering is suppressed, answers must be sought elsewhere. Clinical observations and animal research can give indications which are subject to some uncertainty.

3.3 Active Surface Rewarming

The objective of rapid surface rewarming is to introduce heat into a hypothermia patient at a very high rate. This seems to be intended to accomplish the following:

1. Minimize the time the victim is in the hypothermic state.
2. Quickly revitalize a weakened and irritable myocardium.
3. Avert afterdrop by overwhelming the temperature gradients within the victim's body by a massive infusion of heat.

Obviously active surface rewarming can be accomplished at a range of heat input rates. A widely recommended method is trunk immersion in hot water up to 42°C (Jessen and Hagelsten, 1972). Water heated thermal blankets (Cooper and Ross, 1960), plumbed garments (Webb, 1973) and simple heating pads or hot water

baths could all be considered active surface rewarming but caloric input can vary by an order of magnitude.

Rapid rewarming by hot bath, according to Mills (1976), may cause, "tissue liberation of acid end products of metabolism and the sudden end result of metabolic acidosis may result in death by ventricular fibrillation...."

Hypotension may result (Keatinge, 1969) from the inability of the reduced blood volume to fill blood vessels which are suddenly dilated as a result of active rewarming measures. But according to Golden (1973) it is, "unlikely to have fatal consequences in immersion victims, as the duration of exposure is usually insufficient to permit the occurrence of major physiological adjustments of cirulatory fluid volume."

Weyman, et al., (1974) provide data supporting active rewarming. In studying 39 cases of hypothermia, some complicated by underlying disease, they conclude, "In our experience, neither a decreased core temperature, as manifested by the onset of ventricular fibrillation, nor shock resulted from active rewarming." They further indicate, "that the response to rewarming, as measured by the rate of rise in temperature, depends on the underlying metabolic state of the patient rather than the method of rewarming." Their final recommendation is:

> "In patients in whom the initial temperature is close to the threshold for ventricular fibrillation or in patients in whom response to passive and active rewarming is unsatisfactory, a more efficient method of rewarming is necessary. It has been shown that immersion in warm water at 104° to 108°F (40° to 42.2°C) offers such an alternative."

Tansey (1973) cautions that:

> "The rate of restoration of core temperature must be carefully balanced between the risk of anoxic damage from too brisk an increase in tissue oxygen requirement before circulation is improved and the risk of vascular collapse from the critical afterdrop in core temperature associated with the restoration of peripheral blood through cold deeper layers of subcutaneous tissue."

The major risks of hypoxia, metabolic acidosis, and hypotension may, according to Ledingham and Mone (1978), be controlled with oxygen and intermittent positive-pressure ventilation, sodium bicarbonate, and the administration of warm intravenous fluid.

3.4 Conclusions

When one examines the literature describing slow rewarming, it becomes apparent that the authors are usually considering a situation where the patient is allowed to rewarm himself spontaneously using the body's metabolic processes alone or in combination with a small amount of rewarming supplied by the environment. Rapid rewarming, on the other hand, generally connotes the application of external heat to the patient's body. Just as there is some variation in the details of the slow rewarming, fast rewarming encompasses quite a variety of protocols. The basic factor in common among the various rapid methods is that external heat is applied to the body. However, the rates of temperature rise vary considerably from a fraction of a degree per hour to several degrees per hour. Techniques that vary from body immersion in a tub of hot water to the application of hot water bottles and warm blankets create a wide range of rates of heat input to the body.

As Fernandez, et al., (1970) point out, many of the patients who were warmed with a method called "active" still required over 24 hours to rewarm. This rewarming regimen is clearly in a different class from warming in a tub of water or using a hyperthermia blanket with a total rewarming time under 8 hours.

Active rewarming is said to cause peripheral vasodilatation with resultant afterdrop and possible hypotension. However, quite a different situation results if heat is applied to the thorax with the periphery being allowed to remain cold and vasoconstricted until the central part of the cardiovascular system has warmed sufficiently to supply the metabolic needs and perfusion requirements of the extremities. Some advocates of fast rewarming specifically recommend that the arms and legs be allowed to remain cool.

Many examples of active rewarming in the literature appear to be actively rewarming both the central and peripheral vasculature. Under this circumstance, the periphery can vasodilate returning cold blood to the heart, causing accentuation of afterdrop, possible cardiac arrhythmia, and possible hypotension and shock. There does not seem to be experimental or clinical proof that properly applied rapid rewarming is dangerous any more than there is clear evidence that slow rewarming is dangerous.

Another factor that contributes to the difficulty in resolving the debate between advocates of fast and slow rewarming therapies is the fact that much of the clinical evidence involved is based on cases where the patient is suffering multiple medical problems such as drug overdose or acute alcohol intoxication in addition to hypothermia.

Because of the complexity of man's responses to cold exposure and because of the ambiguity of the meaning of the terms "rapid rewarming" and "slow rewarming", it is difficult to debate their merits in general terms. When specific rewarming protocols are proposed for application under specified conditions, discussion is possible. The remainder of this report addresses specific treatments for profound hypothermia.

4.0 PERITONEAL IRRIGATION

The desirability of core rewarming has been established in the earlier sections of this report. Since peritoneal dialysis is a fairly simple procedure commonly used in the hospital environment, a heated peritoneal lavage presents a means of introducing heat directly to the body core of a hypothermia victim. The procedure does not appear to have been widely used for treatment of hypothermia. Its use was reported by Lash, et al., (1967), Grossheim (1973), and Jessen and Hagelsten (1978) for treating frostbite and hypothermia patients. A case of rewarming by pleural irrigation using 40°C physiological saline is described by Linton and Ledingham (1966).

4.1 Therapy Description

The peritoneal cavity contains the liver, stomach, intestines, kidneys, pancreas, spleen, gall bladder, urinary bladder, inferior vena cava and other structures. This cavity is separated from the pleural cavity (which contains the lungs and heart) by the diaphragm. The peritoneal dialysis procedure involves inserting a large needle through the abdomen into the peritoneal cavity. A liquid dialysate is inserted directly into the cavity and allowed to bath the surfaces of the organs and the highly vascularized mesentery contained therein. When used for rewarming, the dialysate is heated above body core temperature before its insertion into the peritoneal cavity. It is left there until it has surrendered most of its excess heat to the surrounding organs, the inferior vena cava (with its blood supply returning directly to the heart), and the heart and lungs (by conductive heat transfer through the diaphragm). Then the dialysate is removed and replaced by a fresh, warm supply. Dialytic detoxification is accomplished through the diffusion of the toxic substances from the body into the dialysate which is periodically replaced to maintain a large concentration gradient and diffusion rate.

4.2 Advantages and Risks

A basic advantage of rewarming by peritoneal irrigation is that the procedure is fairly common and most hospitals could easily assemble the few items needed to rewarm a hypothermia patient. A distinct therapeutic advantage

is the central application of heat. The greatest value of rewarming by peritoneal irrigation would be when dialysis is indicated, as with drug overdose. The dialysate could both supply heat and remove toxic substances.

Jessen and Hagelsten (1978) list the following as additional advantages of peritoneal dialysis:

1. Rewarming is rapid because of the large heat capacity of the liquid dialysate.
2. It facilitates correction of electrolyte imbalances, particularly hypopotassemia occurring late in rewarming.
3. It does not interfere with the concurrent treatment of other symptoms.
4. The treatment itself does not pose severe risk to the patient.

They base a conclusion that, "peritoneal dialysis is a better means of rewarming than immersion in hot water ...", upon their clinical experience with a rewarming from a 23.4°C core temperature in which the patient did not survive. Two and one-quarter hours in a 40°C bath raised core temperature to only 24°C. This was followed by 2.33 hours of peritoneal dialysis with 5 l/h of 40°C dialysate which raised core temperature to 30.2°C.

Direct warming within the peritoneal cavity offers the advantage of revitalizing the liver. This allows it to resume its functions of detoxification and conversion of lactic acid to glucose.

There is little danger in introducing a large gauge needle into the peritoneal cavity. Viscera tend to be pushed aside with little chance or perforation. Peritonitis and subsequent adhesions are the primary risks of using peritoneal irrigation as a core rewarming procedure. The incidence of these complications is minimal in a hospital.

4.3 Contraindications

Probably the only time when peritoneal irrigation could not be used as a rewarming technique would be if the patient were also suffering from massive trauma of the peritoneal cavity. Under this circumstance warm irrigation during direct surgical exploration would be a logical substitute.

4.4 Guidelines

Core rewarming by peritoneal irrigation should follow the same procedures as the technique applied for dialysis alone except that the dialysate should be warmed to a higher temperature. According to Doolittle (1977) a dialysate temperature of 40.5° to 42.5°C is satisfactory. The rate of heat input can be increased by changing the dialysate more frequently than is usual for dialysis alone. The time interval for exchanging dialysate should be determined by measuring the temperature of the fluid being removed and the victims core (rectal) temperature. A reasonable approach would be to begin with a short exchange interval and increase it until the removed dialysate is found to have lost much of its heat in relation to the core temperature. Detailed guidelines to maximize the therapeutic effect should consider the effects of the dialysate-victim temperature gradient on heat transfer rate and the capacity of dialysis equipment to remove and replace the dialysate.

Jessen and Hagelsten (1978) describe the use of a two-catheter dialysis system with suction at the outflow to achieve high throughput rates (12 l/h). The possibility exists to simplify the procedural aspects of the rewarming therapy by developing equipment to allow the continuous heating and circulating of dialysate through a closed-loop system. This would also reduce the dialysate volume needed to accomplish rewarming, but would disallow dialytic detoxification and electrolyte adjustments.

Jessen and Hagelsten (1978) suggest that hypopotassimia may be treated "automatically" by using a dialysate with a potassium concentration," corresponding to the normal serum-values." This remark seems unnecessary since Guyton (1976), in discussing a "typical dialyzing fluid," indicates that the concentrations of sodium, potassium, calcium and magnesium are normally matched to those found in plasma.

4.5 Conclusions

Core rewarming of hypothermia victims by peritoneal irrigation is a feasible technique that has been successfully used in the hospital. However, one would not expect to see it used as a first-aid measure. The major problems are the difficulty in carrying out a sterile procedure in the field as well as the necessity of a certain amount of diagnosis by an EMT to determine if the severity of the victim's condition warrants the use of peritoneal irrigation.

Probably the main use of peritoneal irrigation for core rewarming is when dialytic detoxification is necessitated as by drug overdose. If the occurrence of hypopotassimia in rewarming is ubiquitous then the attractiveness of peritoneal dialysis would be considerably enhanced.

The procedure does have the advantage that the equipment required for its application is minimal and is readily available at any hospital. Because of the lack of reliable data describing the rewarming effectiveness of peritoneal irrigation no conclusion may be drawn concerning its attractiveness when compared with other therapies. It might be speculated that higher rates of heat input to the core could be achieved with the liquid dialysate than is possible with inhalation therapy (discussed in Chapter 7). A conjecture involving trunk immersion in hot water is more difficult particularly in the context of the clinical experience reported by Jessen and Hagelsten (1978).

5.0 INTRAGASTRIC AND INTRACOLONIC BALLOONS

Heat may be transferred to the body core by intragastric and intracolonic balloons into which warm fluid is pumped. Both approaches supply heat through structures (stomach and large intestine) which are located in the peritoneal cavity. The use of balloons allows the closed-loop circulation of a fluid with high specific heat and which need not be bio-compatible.

5.1 Therapy Description

Both intragastric and intracolonic balloons have been used to transfer heat into or out of the body. Khalil and MacKeith (1954) first described an intragastric balloon for both raising and lowering body temperature. They particularly discussed rewarming advantages, including heating the liver to stimulate metabolic processes. Bernard (1956) described an apparatus for the induction of hypothermia and rewarming using an intragastric balloon. Later Khalil gave details for the construction of intragastric apparatuses for cooling dogs (1957) and for cooling humans with a 1.5 l balloon (1958). A double lumen tube transported fluid to and from the balloon. A provision was made for negative pressure removal of water from the balloon to keep its size constant in the stomach. In 1966, Moss described the use of an intragastric balloon for cooling dogs.

Intragastric balloons have been commercially available although their normal use is for cooling the gastric mucosa to control bleeding due to ulceration. No evidence has been found that this technique has been used to rewarm hypothermia patients.

Intracolonic balloons have been used for controlling the body temperature of animals (Ross, 1954; Cooper and Ross, 1960); but no evidence has been found of the use of the technique in humans. Rewarming by intracolonic balloon would have effects similar to peritoneal irrigation (discussed in the previous chapter) without the risks due to penetrating the peritoneum.

Warm enemas would be another alternative with a similar effect. No literature was found describing the use of warm enemas for rewarming from hypothermia. The use of warm enemas would necessitate larger amounts of fluid than the closed-loop balloon systems and the fluid would have to be bio-compatible.

5.2 Advantages and Risks

Intragastric or intracolonic balloons have the distinct advantage of supplying heat to the core of the body without penetrating the skin. Due to the fact that heat is carried by a fluid rather than a gas, more heat can be supplied to the core via an intragastric balloon than by hot gasses used in inhalation therapy. In addition, the rate of exchange of the rewarming medium is completely under external control.

The intragastric balloon might have an advantage over the intracolonic balloon or enemas in that heat is being supplied closer to the heart, even if the intracolonic balloon were inserted to the transverse colon.

The same advantages due to the stimulation of liver function attributed to peritoneal irrigation (section 4.2) pertain to the intragastric and intracolonic balloons as well.

The use of balloons inserted into the gastrointestinal tract from either end is generally without serious risk. At temperatures below 28°C, however, the heart is usually sensitive to mechanical irritation and the stimulation associated with the insertion of an intragastric balloon could trigger ventricular fibrillation (Hegnauer, et al., 1951). Recently Ledingham and Mone (1978) indicated that they had not observed reflex slowing of the heart precipitating ventricular fibrillation in 38 hypothermia patients treated with endotracheal intubation.

No outstanding danger is recognized in the use of intracolonic balloons or warm enemas for core rewarming. However, the membrances of the colon are thin and traumatic injury is possible, particularly with elderly patients. A potentially significant procedural problem is achieving and maintaining the deep colonic penetrations, desirable for deep core rewarming, against peristaltic attempts to eject the balloon.

5.3 Contraindications

Trauma or surgical intervention involving the gastrointestinal tract would be the primary contraindication to using intragastric or intracolonic balloons for core rewarming. If experimental work proves susceptibility of the heart to mechanical irritation during balloon insertion, use of

the intragastric balloon might be restricted to temperatures above an experimentally determined level. Because of present uncertainties, an intragastric balloon probably should not be used at temperatures below 28°C.

5.4 Guidelines

In view of the recommendations of Doolittle (1977) of dialysate temperatures of 40.5° to 42.5°C for direct peritoneal irrigation, an intragastric or intracolonic balloon may be heated by water up to 40°C with impunity. Khalil (1958) suggested rewarming patients using water heated to 40°C.

Because of the possibility of heart irritability at or above temperatures of 28°C extreme care should be exercised in inserting the intragastric balloon assembly.

5.5 Conclusions

Intracolonic, and more particularly intragastric balloons, offer some promise as a core rewarming therapy without the requirement for sterile procedures attendant to peritoneal irrigation. The literature survey does not reveal that the procedures have received much clinical or experimental attention as rewarming therapies. Further experimental work is warranted since appreciable heat may be conveyed to the body core by these rewarming therapies.

6.0 EXTRACORPOREAL BLOOD REWARMING

Cardiac bypass in open heart surgery has become an almost routine surgical procedure during the past several years (Ionescu and Wooler, 1976). Cooling is used to protect tissue from anoxia during cardiac standstill. External rewarming of the blood and body follows surgery before the patient is removed from the bypass equipment.

6.1 Therapy Description

Extracorporeal blood rewarming may be accomplished using several circuits as discussed by Cooper and Ross (1960). Systemic heparinization is required. The simplest extracorporeal circuit (involving the least surgery) uses large bore cannulas to remove blood from the femoral veins. It is oxygenated, warmed, and returned to the circulatory system via the femoral artery. According to Truscott, et al., (1973), a disposable oxygenator and heat exchanger may be primed with only 70 ml of a solution containing no blood.

As a rewarming therapy, the objective is to return warm blood directly to the heart such that this organ may be heated sufficiently to meet the cardiac output demands of the rest of the body as rewarming progresses. The method has been used successfully to rewarm victims of accidental hypothermia (Davies, et al., 1967; Truscott, et al., 1973). The case reported by Truscott and his associates involved a woman cooled to 22°C with ventricular fibrillation and respiratory failure.

6.2 Advantages and Risks

The primary advantage of extracorporeal rewarming is that the heart is rewarmed almost directly by heated blood. This has the immediate advantage of reducing the irritability of the weakest link in the hypothermic "chain". As long as the heart is rewarmed prior to rewarming of other body tissues shock due to depressed cardiac function is unlikely. While peripheral vasodilatation should be controlled, hypovolemic shock due to peripheral vasodilatation can be compensated for more successfully by a warm heart. According to Barnard (1956) extracorporeal blood cooling (and presumably rewarming) is faster than surface

treatments as a means of controlling body temperature. The procedure produces almost ideal rewarming conditions as mentioned above.

The risks of extracorporeal rewarming primarily relate to the use of extracorporeal techniques rather than its effect on hypothermia recovery. Complications and risks are associated with the need to heparinize the patient, damage to red cells by the bypass equipment, arterial cannulation, and others. In addition there is always the problem of blood hemolysis and of air leaking into the circuit. The requirement for arteriovenous punctures involve risks of vessel destruction, release of plaque material, and ischemia due to spasm of the vessel. In the proper hands, the procedure can be reasonably safe but it is a complicated, highly invasive procedure that requires special training and equipment.

6.3 Contraindications

Specific contraindications for extracorporeal blood rewarming include all conditions which would be compromised by the use of anticoagulants such as heparin. These include bleeding, both internal (e.g., ulcers) and external (e.g., trauma).

6.4 Conclusions

Extracorporeal blood rewarming is a procedure that has been used successfully in the treatment of accidental hypothermia (Davies, et al., 1967). There is little question that the technique can be used quite successfully in the hospital environment by properly trained personnel. It involves a significant surgical procedure; requiring a high level of medical competence. Consequently, hypothermia rewarming by extracorporeal bypass should be considered only in a hospital and only as a final lifesaving measure in cases that do not respond to more conservative methods.

7.0 INHALATION THERAPY

The desirability of rewarming the core of a hypothermic patient has been discussed repeatedly in this report. Most core rewarming methods (such as extracorporeal circulation, peritoneal irrigation and direct warming of the heart after thoracotomy) are either major surgical procedures or are, at best, invasive. Recently, groups working independently in England, Australia, and Canada have advocated core rewarming by the inhalation of hot moist gasses. Emphasis is placed on the suitability as a first-aid treatment although the techniques differ somewhat.

7.1 Therapy Description

Lloyd originally mentioned inhalation therapy in The Lancet (1971) and later Lloyd, et al., (1972) and Lloyd (1973) described an apparatus in some detail and reported its use on seven hypothermic patients. The device is based on the well known exothermic reaction of soda lime and carbon dioxide. A canister of soda lime is pre-heated by "firing" CO_2 cartridges through it. The heated canister then serves to heat and humidify oxygen breathed through it using a to-and-fro Water's anesthesia circuit. Expired carbon dioxide continues to heat the soda lime, and the incoming oxygen supply.

Shanks and Marsh (1973) described the rewarming of a patient on a Bird respirator using heated water-bath humidifier. They used hospital equipment and did not emphasize the technique as a first-aid measure for use in the field.

More recently Hayward and Steinman (1975) and Collis, et al., (1977) have reported experiments specifically aimed at developing suitable first-aid procedures. They used Bennett Cascade humidifiers to humidify oxygen and warm it to be inspired at temperatures from 40° to 45°C and 43° to 48°C, respectively. Hayward and Steinman (1975) compared inhalation therapy to wholebody rewarming in a hot whirlpool bath. They indicated no significant difference in the afterdrops of rectal and tympanic temperatures with the two therapies; but the level of significance of this conclusion is not given.

Collis, et al., (1977) compared these two therapies with three others - electric heating pads, inhalation therapy combined with heating pads, and

shivering. In a comparison based on afterdrop in rectal and tympanic temperatures, the only difference found to be statistically significant at the 0.005 level was between shivering and oxygen inhalation therapy. They conclude inhalation rewarming to be, "an effective emergency therapy for accidental hypothermia." No indication is given of what additional differences might have been significant at more commonly-used levels of significance, say 0.01 or 0.05.

Most recently Marcus (1978) studied inhalation rewarming at "44±1°C from a Marshall Spalding water bath humidifier...." He used an open loop system with the subjects inspiring air. Inhalation rewarming was compared to three other therapies - hot bath (arms out), piped suit, and spontaneous (shivering). He reports no significant differences in the afterdrop of auditory canal temperatures among the four rewarming therapies; but the level of significance of this conclusion is not given. Some differences were found in the rates of restoration of heat. Hot bath rewarming was found to be significantly quicker than the piped suit at the 0.01 level and the piped suit was found to be significantly quicker than inhalation therapy and spontaneous rewarming at the 0.001 level. He concludes that, "in the absence of some powerful method of central rewarming, the most effective way of raising a depressed core temperature is by actively applying heat to the skin, and inhalation rewarming should not be employed if it entails a delay in a patient reaching some means by which this can be carried out." He continues, "[if] the patient is insulated within a heated shelter, inhalation of hot air will not be significantly beneficial, it will not prevent an afterdrop...."

7.2 Advantages and Risks

Breathing hot moist gas offers several advantages as a first-aid measure:

1. The technique warms lungs and heart first.
2. The procedure offers a curtailment of respiratory heat loss.
3. Thermal stimulation of ciliary activity is afforded.
4. The equipment is simple and inexpensive.
5. The equipment is easy to transport.
6. Lloyd's device requires no external power source.

It should be pointed out that the techniques described in Lloyd's and Hayward's groups require the patient to be breathing spontaneously. The rate of heat input is a strong function of respiratory minute volume. The Lloyd apparatus and the Hayward system can be "bagged" to apply forced ventilation. The Shanks system is, of course, attached to a respirator.

Oxygen Inhalation Therapy

Collis, et al., (1977) cite as an advantage of oxygen inhalation therapy an, "increase in coronary arterial oxygenation." This is expected to forestall, "ventricular fibrillation from a limitation of myocardial energetics...." It may also be expected to counter the tendency toward anoxic pulmonary edema.

The specific gas mixture to be used in inhalation rewarming is an important part of the overall therapeutic formulation. The use of 100 percent oxygen may not be desirable. Negovskii (1962) summarizes some evidence supporting this proposition. He cites a histological examination, by Romanova (1956), of the brains of 20 dogs. Pure oxygen was used to resuscitate the dogs after being clinically dead for 5 minutes. Oxygen therapy was continued after the resuscitation of some of the dogs. Fifteen died within the first 5 days and 5 were sacrificed 14 to 90 days after resuscitation. In the 15 animals dying within the first 5 days, characteristic findings included, abundant small hemorrhages in all divisions of the brain, swollen vascular endothelium, and separation and homogenization of the walls of some vessels. These changes were not seen to such a pronounced extent in animals not receiving oxygen, even after longer periods of clinical death. Hemorrhages and changes in vascular walls were also found in the heart, lungs and liver. Circumscribed changes in the brain, including structural disturbances, death of nerve cells and scar formation, were found in animals dying in a moderately prolonged period of time only when oxygen therapy was given. He suggests that oxygen concentration during artificial respiration should not exceed 30 to 40 percent. He cites Dembo (1957) who suggests that oxygen therapy is best performed at a concentration of 50 percent and Swann (1950, 1951) indicating that, "very small doses of oxygen are needed for resuscitation (3 to 4 percent in acute anoxia)." The single exception of carbon monoxide poisoning is mentioned.

Alexander (1945) summarizes the results of oxygen inhalation after the cooling of 4 subjects at Dachau:

"Oxygen inhalation, however, had no effect either upon respiration or upon heart action. The authors [Holzloehner, Rascher, and Finke (1942)] feel that the markedly bright color of the arterial blood [observed in other human experiments] made it unlikely that additional oxygen would have any beneficial effect."

The oxygen therapy was examined because of, "the clinical picture of dyspnoea, with formation of foam at the mouth...reminiscent of early oedema of the lungs...."

Hedley-Whyte, et al., (1976) observe that, "Pure oxygen probably causes [in normothermic patients] marked depression of ciliary activity. Mucus flow is impaired. Tracheitis, as noted by bronchoscopy, is apparent after only six hours of 100% oxygen."

Thermal Considerations of Inhalation Gas Mixture

A wide variety of physiologically acceptable gas mixtures could be used in inhalation rewarming. Constituent gasses with high molar heat capacities will transport more heat per breath at a given temperature than those with lower molar heat capacities. Molar heat capacity is largely determined by the number of atoms in a molecule of the gas. For example, triatomic gasses have higher molar heat capacities than diatomic gasses. Unfortunately, the effects are not large and the opportunities to use gasses with complex molecules are limited due to the requirement for physiologic acceptability. The water vapor in the inhalation gas carries considerably more heat than the warm gas itself.

Inhalation Therapy With a CO Gas Fraction

A gas mixture containing some CO_2 might be expected to offset the depressing effects of profound hypothermia on respiration thereby enhancing the early thermal benefit to be derived from inhalation rewarming. In experiments reported by Morrision, et al., (1978) volunteers with rectal temperatures depressed by 2°C were given inhalation therapy at 44±1°C with two different gas mixtures. One rewarming was performed with air, the other with air combined with a variable amount of CO_2 adjusted to maintain respiratory minute volume at 45 to 50 l. With air-inhalation rewarming, respiratory minute volume averaged 21 l over the test period. The difference between the afterdrops observed with the two therapies was not significant at the 0.05 level. However, the rates of increase in both rectal and tympanic temperatures were greater at

this level of significance with the CO_2-induced hyperventilation. This was asserted by one of the researchers (Hayward, personal communication, June 15, 1978) to support the proposition that inhalation therapy affects the rate of core rewarming since a variation in the respiratory minute volume produced a corresponding significant variation in core rewarming rate. Even so, no claim is made for the efficacy of performing inhalation rewarming of victims of profound hypothermia using a CO_2 gas fraction. Indeed, one would expect CO_2 inhalation to accentuate the acidosis described in section 2.4 of this report. Whether or not a small CO_2 gas fraction might, on balance, improve the chances of successful resuscitation from profound hypothermia remains unknown.

Therapeutic Effects of Humidification

As was indicated earlier, the water vapor in the inhalation gas carries much more heat than the gas itself. Much of this heat content is due to the 540 cal/g latent heat of vaporization of water. But for this heat to be imparted to the lung tissue the water vapor must condense in the lung. Shanks and March (1973) estimated that the heat input to the body is less than 10 kcal/hr at a respiratory rate of 10 l/min. However, it should be recognized that respiratory heat loss could be as high as 13 kcal/hr in cold weather. This heat loss could be a significant portion of the patients depressed metabolism and would be prevented by the use of inhalation therapy. Thus the patient's heat balance would be improved by 23 kcal/hr as a result of receiving inhalation rewarming.

As discussed in Section 2.3 there may be pulmonary congestion of some severity present in the victim of profound hypothermia. Unfortunately some effects of water vapor inhalation may depend upon the nature of this congestion. If the congestion is due to pulmonary edema, the condensation of additional water may compound the problem. Collis, et al., (1977) cite Toung, et al., (1970) describing the ability of the respiratory tract to transfer large amounts of fluid into the vasculature. The ability to absorb fluids is predicated upon a favorable balance between osmotic and hydrostatic pressures and upon the normal functioning of blood vessel membranes. The existence of pulmonary edema is prima facie evidence of the failure of one or more of these elements to preserve the fluid contents of the vasculature. Therefore, there is reason to doubt the reliability of the fluid transfer mechanism to contend with condensing inhalation vapor in addition to pulmonary edema existing in a victim of profound hypothermia.

The preceding situation contrasts markedly with that of the alternative source of pulmonary congestion discussed in Section 2.3. When the congestion results from the accumulation of mucus and other secretions due to cold-inhibition of ciliary activity, inhalation rewarming may have desirable non-thermal therapeutic effects, in addition to the direct thermal stimulation of ciliary activity. No treatment recommendation has been found for this specific condition. However, the treatments recommended for related conditions suggest that humidification may be helpful. Potter (1976), in prescribing treatment of mucopurulent bronchitis (inflammation of the bronchial tree accompanied by a marked exudative process), states: "Ancillary measures include nebulization therapy to add water to the secretions for the purpose of reducing their viscosity." He observes that there is continuing debate over the usefulness of the nebulization therapy with some contending that it does not reach the deep areas where it is most needed. It should be noted that inhalation rewarming deposits water by condensing vapor on the relatively cool lung tissues. This may reach deep tissues better than would suspended water particles. Potter concludes that, "The nightly use of a unit which generates cold mist has also been found to be at least of subjective value, but the particle size is such that only the upper airways are benefited." Bouhuys (1977) argues that, "water and mucus do not mix to form a homogeneous liquid, and addition of water, therefore, does not liquify mucus." He cites experiments by Parks, et al., (1971) which indicated that, "exposure to mists...caused no change in tantalum clearance rate in dogs." Bendixen, et al., (1965) describe mist therapy as, "often effective in short-term treatment of laryngeal edema or in cases of very thick tracheobronchial secretions." They also indicate that, "Large amounts of secretions are produced by most patients with chronic pulmonary disease. Treatment with abundant humidity...is required." Ashbaugh (1971) in prescribing care for tracheostomy patients observes that, "bronchial secretions become more viscous and tenacious, leading to atelectasis and infection. Both prevention and treatment of this condition depend on the maintenance of a constant source of moisture in the breathed air or oxygen."

7.3 Contraindications

The major contraindication for inhalation rewarming would be traumatic injury to the face. In this circumstance other first-aid measures might take precedence. However, the technique can be applied by tracheostomy but the temperature must be more carefully controlled.

It is not presently clear if pulmonary edema of some severity might be a contraindication for inhalation rewarming. The notion is sufficiently plausible to warrant further investigation.

7.4 Guidelines

Heated gas at the mouth should be limited to an inspired temperature of less than 45°C to protect the mouth. A conscious patient can complain of heat discomfort but when used with a patient less than fully conscious instrumentation is probably needed to monitor temperatures delivered by heat humidifiers. The temperature of the soda lime canister described by Lloyd can be monitored by touch, as 45°C is about the maximum that the bare hand can withstand.

Collis, et al., (1977) point out that distilled water is routinely used in clinical humidification and was used in their inhalation device.

The equipment should be discarded after use or thoroughly sterilized between uses to preclude the spread of communicable diseases.

7.5 Conclusions

It has not been clearly established that inhalation rewarming alone can prevent or significantly reduce afterdrop in a seriously cooled patient. Experimental results based on mildy cooled volunteers are conflicting. The positive findings of Hayward and Steinman (1975) and Collis, et al., (1977) are supported by a larger base of experimental data than are the negative findings of Marcus (1978). The hot-bath therapy formulations used as bases of comparison for inhalation rewarming by the Canadians (arms and legs in) and by Marcus (arms out, legs in) were less likely to prevent afterdrop than a formulation with arms and legs out of the bath. Therefore, their results with that particular therapy may be regarded as pessimistic bounds on its potential performance when applied only to the body trunk.

As a practical matter, the question of gas mixture reduces to a choice of oxygen concentration. Based on the experience of Negovskii (1962) and others, the oxygen tension should be no larger than can be expected to be fruitful. Air may be sufficient for most inhalation rewarmings from immersion hypothermia due to its oxygen partial pressure at low altitudes and based on the observations of Holzloehner, et al., (1942). An open loop, air-inhalation circuit is certianly the simplest from a sterilization point of view; but it may require greater

heating capacity and may produce inhalation temperatures which are more difficult to control than a closed loop system.

Little can be said concerning the magnitude of the problems arising from applying inhalation rewarming to victims of profound hypothermia who exhibit pulmonary edema. Further experimentation with animals in profound hypothermia and systematic clinical observations are needed to establish the nature and severity of any pulmonary edema and the interaction of inhalation rewarming with this condition. There are, of course, treatments for the edema which could be used to clear the way for the application of inhalation rewarming. These treatments are generally directed toward the cause of the edema, which is not presently known. Furthermore, this treatment of edema could involve significant delays in the initiation of inhalation therapy. During this time the patient should, as always, be handled so as to minimize afterdrop during the treatment of pulmonary edema. Rewarming could be initiated with another therapy during this time with inhalation therapy added when the pulmonary congestion has been cleared.

Inhalation rewarming provides direct thermal stimulation of ciliary activity which would tend to clear congestion from the pulmonary tree. Although there are dissenting views, the majority opinion seems to be that humidification tends to liquify tenacious congestion and mobilize it for transport.

The inhalation rewarming of a victim of profound hypothermia (with depressed respiration) could be accomplished in two ways. The simplest approach would be to allow natural respiration to cause heat to be infused at a very low rate. A more aggressive approach would involve bagging the inhalation gas to elevate respiratory minute volume and the resulting heat transfer. This could tend to offset existing acidosis, perhaps to the point of precipitating alkalosis which, in turn, could be corrected by increasing the CO_2 content of the inhalation gas mixture. This more aggressive approach would be less simple and could necessitate relativey complicated monitoring of variables. The potential also exists for bagging to produce mechanical irritation of a weakened myocardium increasing the tendency toward fibrillation.

It is not necessary that inhalation rewarming be as effective as torso immersion in a hot bath for it to be useful as a first-aid treatment. The inhalation of warm, moist gasses prevents respiratory heat loss, infuses a small amount of heat into the deep body core near the heart and stimulates ciliary activity. This alone is probably sufficient justification for its use.

8.0 DIATHERMY

The word diathermy comes from the greek dia, meaning through and therme, meaning heat. It has come to include several varying ways to transmit heat. Lehmann (1971) defines it, for therapeutic purposes, as "deep heating". He discusses three modalities which are used for therapeutic purposes -- ultrasonic, short-wave and microwave diathermy. They share the characteristic of accomplishing heating by conversion. This implies that various types of energy are transmitted through superficial tissues to deeper ones where they are converted into heat.

8.1 Descriptions of the Therapies

Ultrasonic Diathermy

Ultrasonic diathermy utilizes high frequency acoustic vibrations (0.8 to 1.0 MHz) propagated in the form of longitudinal compression waves. The heating in response to therapeutic intensities (1 to 4 watts/cm^2) derives from tissue particle movement as a result of the wave propagation. These waves will not propagate through air, so physical contact with the transducer is required. Wave intensity diminishes exponentially with depth in a homogeneous tissue. According to Lehmann and Johnson (1958), the ultrasound absorption per unit of tissue thickness in fat is about twice that in muscle and in bone is over 10 times that in muscle. Reflection can occur at the interface between materials of different acoustical impedances. This effect is particularly pronounced with bone and surgical implants. Carstenson, et al., (1953) and Piersol, et al., (1952) showed that absorption and conversion to heat occur primarily in the tissue proteins. According to Schwan (1958) relatively little energy is converted into heat in the subcutaneous fat and much more is converted in the musculature. One half of the energy available at the fat-muscle interface is available 3 cm into the muscle.

Short-Wave Diathermy

Short-wave diathermy utilizes high frequency current (13.56 to 40.68 MHz) to establish a current within the "patient circuit". This current is tuned to resonance, usually with a variable capacitor, by maximizing a power indication. The therapy may be applied either with a capacitance device or an

induction device. According to Lehmann (1971) short-wave diathermy produces heating at a tissue depth which is, "between that produced by superficial heating agents... and [that produced by] low frequency microwave or ultrasound...." He cites short-wave diathermy as, "an efficient agent to heat musculature up to a depth of 1 to 2 centimeters." Swan, et al., (1955) report the regular use of a standard diathermy unit with the induction coil wrapped around the pelvis for rewarming surgery patients. Their data shows the method to be as rapid for rewarming as immersion in water at 45°C. They also state, "Occasionally, for reasons not apparent, the diathermy has also proved inefficient." In 1952 Bigelow, and his associates described a series of experiments on dogs and monkeys with the specific objective of developing, "a safe radio-frequency resuscitation procedure requiring only a small portable radio-frequency generator and power supply with a simple technique of application." The complete summary of the paper follows:

"1. A radio-frequency induction cable technique has been developed which has successfully rewarmed dogs and monkeys from near-lethal hypothermia of 21°C to normal body temperature.

2. The short wave therapy range was found most effective for heating the deep tissues of large animals.

3. Spacing of coils from body was the most critical rewarming factor. Using one-half inch spacing, adequate heat generation was obtained without burning the subcutaneous tissue.

4. A frequency of 13.56 megacycles and an insulation of one-half inch was finally selected as the safest and most effective combination and became a standard technique.

5. A final series of animals was resuscitated by this technique. They all survived with no burns, no signs of vascular collapse, and no post-rewarming change in behavior or intelligence."

Alexander (1945) summarized the results of human rewarming experiments conducted at Dachau employing "short wave" diathermy of the heart.

> "The treatment had no demonstrable beneficial effect. It was likewise considered unsatisfactory because in view of the loss of skin sensation due to the cold, the danger of extensive burns exists even though a physician may exercise constant supervision."

The wave lengths, modes of application and energy levels used were not reported. Equipment available at the time did not permit whole-body treatment of the human subjects. The experiments had been prompted by previous successes with whole-body animal rewarming which, according to Holzloehner, et al., (1942), "...leads to a recovery of the animal with puzzling rapidity."

Microwave Diathermy

Microwave diathermy transmits energy in the form of electromagnetic radiation in the 915 to 2450 MHz range. Heat is created in the tissues as a result of their resistance to the impinging radiation. Guy, et al., (1974) indicated that the longer wave length associated with the industrial, scientific and medical frequency (915 MHz) gave more power transferred to deep tissues than with 2450 MHz. They summarize the deficiencies of 2450 MHz diathermy:

"1. absorption is so great in the muscle layer that the depth of penetration is only 1.7 cm.

2. the severe discontinuity at the fat-muscle interface produces a large standing wave resulting in a "hot spot" in the fat layer one-quarter wave length from the muscle surface.

3. the absorbed power density in the deep tissues varies considerably with fat thickness making it difficult to predict the proper therapeutic level for different patients having a wide variation of fat thickness."

Concerning 915 MHz diathermy they observe, "The penetration and minimal fat heating compare favorably with those of the short-wave diathermy, with the additional advantage that the heating pattern is reasonably uniform in contrast to the undesirable toroidal pattern of the latter." Lehmann (1971) indicates that 50 percent of energy available at muscle surface is available at a 3 cm depth with 900 MHz microwaves while it was available to only a 1 cm depth with 2450 MHz.

8.2 Advantages and Risks

The primary advantage for diathermy rewarming is its ability to heat tissues below the subcutaneous fat layer. This advantage pertains particularly to ultrasound and low frequency microwaves; but short-wave diathermy is also capable of some deep tissue heating. Swan, et al., (1955) point out that rewarming by diathermy is particularly valuable since coils can be positioned

over the pelvis, if necessary, so that manual cardiac compression can be applied to support circulation until a patient can be rewarmed sufficiently for spontaneous defibrillation to take place or until a defibrillator can be used effectively (core temperature above 28°C).

A risk with diathermy, particularly short-wave and microwave, is superficial burns. These can result from application of excessive energy, moisture on the patient's skin, improper positioning of the applicator or movement of it during treatment. The problem is reduced in short-wave diathermy by the use of the induction coil applicator. Advances in ultrasound equipment have provided the capability to detect transducer movements which could increase the transducer load intensity and automatically reduce the system output.

A more difficult problem is the determination of proper treatment dosage. Verbal feedback from the patient is generally used in physical medicine to determine control settings on a diathermy unit. A semiconscious or unconscious hypothermia victim would be unable to provide information about the heating effect of the treatment. Dosage can be approximated by knowing the dosage energy input and dosage time; but the exact distribution of thermal effects will not be known. The therapy will have to be applied conservatively with close monitoring of rectal temperature.

The fact that Swan, et al., (1955) regularly used the technique on surgical patients suggests that the overheating problem can be controlled. They applied the therapy intermittently, two minutes on and one minute off.

8.3 Contraindications

The complete lists of contraindications for the three diathermy modilities are given by Lehmann (1971). The following are contraindictions for both short-wave and microwave diathermy.

1. any question of hemorrhage
2. areas of sensory loss
3. moist dressings or adhesive tape
4. regions of suspected malignancy
5. presence of phlebitis
6. tuberculosis
7. joints with effusions
8. areas of occlusion arterial disease

9. areas containing metallic implants
10. during pregnancy or menstruation
11. on patients with pacemakers.

Additional specific contraindications given for microwave diathermy are the area of the eye and edematous tissue. As indicated in section 2.3 of this report edema may be a consequence of hypothermia. Regarding short-wave contraindications Lehmann (1971) states, "It remains to be investigated whether fluid accumulation in the body cavities, such as the joints and pleural cavity, represent an indication for decreasing the dosage since selective heating of these may occur." Item 2 above addresses the problem of dosimetry which must be solved to permit treatment of unconscious patients.

There are fewer specific contraindications for ultrasonic diathermy, as follows:

1. avoid therapeutic dosage to the eye
2. spinal area after laminectomy
3. hemorrhagic diatheses
4. regions or suspected malignancy
5. area of vascular insufficiency.

Since the eye is contraindicated because of the danger of damage caused by cavitation of the fluid contained therein, it is surprising that pulmonary edema is not listed for the same reason.

8.4 Guidelines

For short-wave and microwave diathermy, wet clothing should be removed and the patient should be thoroughly dried so that hot spots and burns do not develop on the body surface. Terry cloth is usually used with short-wave diathermy to absorb any residual mositure or perspiration and to help space induction heating coils at least 1/2 in from the skin. The latter spacing is recommended to prevent burns. Short-wave therapy by induction coil heating is recommended rather than capacitive heating with large plates.

Since metallic implants or pacemakers are contraindications for short-wave and microwave diathermy, any patient to be rewarmed by these methods should be thoroughly checked for thoracic or hip scars that would suggest implants in or

near the regions to be heated. Also, due to the danger of strong, local induced heating, all metallic objects such as rings or bracelets should be removed prior to treatment.

Ultrasonic diathermy should be applied with a heavy oil as a mediator between the transducer and the patient's skin.

8.5 Conclusions

Because of the potential for delivering significant amounts of heat to relatively deep tissues, diathermy, particularly ultrasonic and low-frequency microwave, should receive serious consideration for possible use as a rewarming therapy.

The primary obstacle to diathermy rewarming is the development of dosimetry guidelines which are effective yet safe in the absence of reliable feedback from the patient. The seriousness of the edema contraindications should be determined. Research is also needed to determine the best way to apply each modality to accomplish core rewarming. Pelvic application has been suggested and would probably advance rectal temperature most quickly; but an application nearer the pleural cavity might produce better therapeutic results. The location of bone structure will differentially affect the performance of the three modalities.

Diathermy requires equipment of some sophistication; but it is commonly operated by technicians. The equipment may be made reasonably portable; but it requires a supply of electric power.

9.0 APPLICATION OF THE THERAPIES

Three points regarding the application of the rewarming therapies considered in this report are of paramount importance. They are the suitability of the therapies for use as first-aid treatments in the field (out of a hospital environment or its equivalent) and their suitability for use either simultaneously or sequentially during the restoration of normothermia. These points are addressed in this chapter.

9.1 First-Aid Treatments

The selection of first-aid therapies for which EMT's are to be trained and equipped involves a tradeoff between the therapeutic advantage of a therapy versus the risk to the patient of receiving the therapy from technicians under the conditions prevailing at the accident site or in route to a hospital environment. This risk should not be confused with a risk to the patient of receiving no treatment at all. This latter risk is involved in the determination of therapy benefit. If quantitative measures could be obtained for the benefit and risk inherent in each of a number of candidate rewarming therapies, the best could be selected by comparing the benefit/risk ratios of the therapies. Ignoring morbidity due to the therapies, this could be formulated as the selection of the therapy (counted $j = 1, 2, \ldots, n$; where there are n candidate therapies under consideration) which maximizes the expression:

$$\left[\frac{\Pr(\text{survival with treatment } j) - \Pr(\text{survival without treatment})}{\Pr(\text{death due to treatment } j)}\right]$$

These probabilities, indicated by Pr, would be difficult to estimate accurately with information presently available. Therefore this report is restricted to a delineation of the views of the authors concerning the suitability of the therapies for use as first-aid treatments.

Of the potential first-aid measures, rapid surface heating by trunk immersion in hot water offers good potential on shipboard or wherever a bathtub is available. Hot water immersion is not likely to be a viable procedure aboard a small boat or rescue helicopter although special plumbed garments or body suits filled with hot water offer a possible alternative. Systems could be developed which utilize the waste heat from the vehicle engine.

Of the therapies which have been examined in this report, the following four may reasonably be performed by EMT's outside of a well-equipped medical facility:

1. Surface rewarming
2. Intragastric balloon
3. Inhalation rewarming
4. Diathermy

Passive slow rewarming, intragastric balloons, inhalation rewarming, and diathermy are rewarming techniques which could be initiated in a small boat, helicopter, or some other small rescue vehicle. Either no equipment is required (as in the case of passive rewarming) or else compact emergency equipment exists or could easily be developed.

Peritoneal irrigation offers some potential for use as a first-aid procedure under reasonably controlled conditions by specially-trained EMT's with special equipment to facilitate monitoring of dialysate and core temperatures and to control dialysate replacement.

Extracorporeal blood rewarming should remain a hospital-based procedure that is reserved for serious hypothermia cases that cannot be rewarmed by more conservative means or that must be used if the patient develops ventricular fibrillation. Indeed extracorporeal blood rewarming should probably be performed only by specially-trained physicians.

9.2 Simultaneous Treatments

Some of the therapies examined in this report should not be applied simultaneously. Diathermy should not be applied in the area while either an intragastric or intracolonic balloon is being used or while the peritoneal cavity is being irrigated. This is particularly the case for ultrasonic and low-frequency microwave diathermy due to the penetration depths they can achieve and their undesirable effects on accumulations of water. It would probably be prudent to withhold short-wave diathermy as well.

There is no immutable reason why inhalation therapy and the intragastric balloon could not be administereed simultaneously. However, because of their requirements to access the body through the pharynx, special equipment may be needed to enable their simultaneous application.

The remaining combinations of pairs of the therapies under consideration are all quite feasible.

9.3 Sequential Treatments

Because treatment is sometimes initiated but not completed in the field, it is reasonable to ask if the initiation of a particular therapy might preclude the later use of another therapy when the victim is transported to a facility capable of a more intense level of care. In short, no specific contraindications have been identified which would preclude sequencing these therapies in any order. It should be noted that this finding does not address the question of the therapeutic desirability of these sequences. The conclusion is only that the therapies may be safely sequenced in any order.

10.0 SUMMARY AND CONCLUSIONS

The literature on resuscitation from accidental hypothermia is split between advocates of fast active rewarming and slow passive rewarming. Survival statistics do not permit a definitive evaluation of the various methods at present. Some experimental evaluations of simple, non-invasive therapies have been performed using volunteers with mild induced hypothermia (rectal temperatures around 35°C). The need still exists for more systematic and comprehensive studies of this type. Of course these studies are limited in that they can not reveal the effects of the therapies when applied to a profoundly hypothermic patient who is unconscious, not shivering, acidotic, subject to hypovolemic shock, and perhaps exhibiting one or more types of edema. Information concerning the performance of the therapies under these conditions can only come from conjecture, clinical observations, and experiments with deeply cooled animals. Conjecture, even when based on good knowledge of fundamentals is the weakest basis for conclusions because of the complexity of the interactions in biological systems. Clinical observations are useful in suggesting lines of scientific inquiry but are a weak basis for conclusions because of their uncontrolled nature. Experimentation with deeply cooled animals is an imperfect source of information, as is experimentation with moderately cooled humans. However, it also should be drawn upon for indications of the safety and efficacy of rewarming therapies before forming conclusions about their suitability for use.

Rapid external rewarming by immersion of only the torso in warm water is commonly regarded as safe and effective. There are obvious physical limitations on its usefulness as a first-aid treatment. A large tub of hot water is not a highly portable item.

When patients cannot be rewarmed by conservative means or when ventricular fibrillation occurs at a body core temperature below 28°C, aggressive measures must be taken. Diathermy, peritoneal irrigation and extracorporeal blood rewarming have all been employed successfully.

Diathermy has good potential for use as a first-aid treatment. It should be investigated further to determine the best modality and protocol for application to safely achieve best core rewarming and organ system revitalization. Its sensitivity to edema as a contraindication must be determined.

Peritoneal irrigation offers limited use as a first-aid treatment. Its best use is when dialytic detoxification is desired as with drug overdose.

Extracorporeal blood rewarming requires surgical invasion which would be very dangerous in the field. The requirement to administer anticoagulants would counterindicate its use in all cases involving trauma.

Rewarming by inhalation of warm humid gasses offers distinct potential as a first-aid treatment since it at least prevents further respiratory heat loss and stimulates ciliary activity. Its interaction with the pulmonary edema possible in profoundly hypothermic patients warrants investigation.

The use of an intragastric balloon offers some potential as a first-aid rewarming treatment. It introduces heat into the core while necessitating less need for sterilization than other invasive treatments. Its clinical effectiveness has not been determined but appears promising.

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