



Virginia Commonwealth University
VCU Scholars Compass

Theses and Dissertations

Graduate School

1989

Efficiency of the Thermal Jacket on the Delivered Temperature of Prewarmed Crystalloid Intravenous Fluid

Danny R. Bowen

Follow this and additional works at: <https://scholarscompass.vcu.edu/etd>



Part of the [Nursing Commons](#)

© The Author

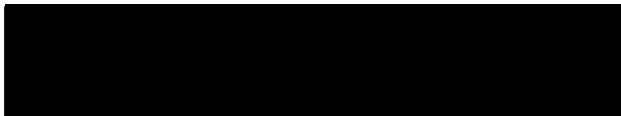
Downloaded from

<https://scholarscompass.vcu.edu/etd/4372>

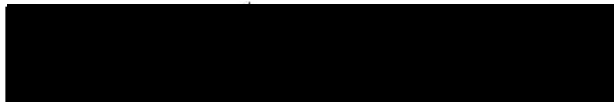
This Thesis is brought to you for free and open access by the Graduate School at VCU Scholars Compass. It has been accepted for inclusion in Theses and Dissertations by an authorized administrator of VCU Scholars Compass. For more information, please contact libcompass@vcu.edu.

School of Allied Health Professions
Virginia Commonwealth University

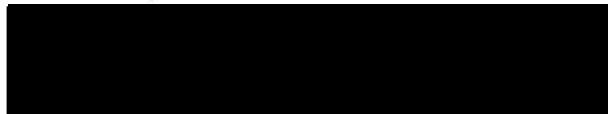
This is to certify that the thesis prepared by Danny R. Bowen entitled
EFFICIENCY OF THE THERMAL JACKET™ ON THE DELIVERED
TEMPERATURE OF PREWARMED CRYSTALLOID INTRAVENOUS
FLUID has been approved by his committee as satisfactory completion of the
thesis requirement for the degree of Master of Science in Nurse Anesthesia.



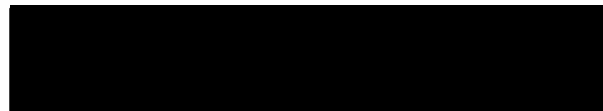
Director of Thesis



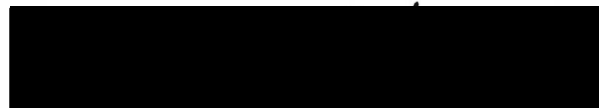
Committee Member



Committee Member



Department Chairman



School Dean

6/14/89

Date

EFFICIENCY OF THE THERMAL JACKET™ ON THE
DELIVERED TEMPERATURE OF
PREWARMED CRYSTALLOID INTRAVENOUS FLUID

A thesis submitted in partial fulfillment of the
requirements for the degree of Master of
Science in Nurse Anesthesia at
Virginia Commonwealth University

By

Danny R. Bowen
Bachelor of Science in Nursing
Ball State University, 1983

Director: John T. Beeston, BS, CRNA
Assistant Professor
Department of Nurse Anesthesia
School of Allied Health Professions

Virginia Commonwealth University
Richmond, Virginia
August, 1989

Acknowledgments

This thesis is dedicated to my wife Penny. Her unselfish love, devotion, and support over the last ten years have been an unending source of strength for me. Being husbandless for most of the past two years, she has been both mother and father to our two children, Brandon and Andrea. Neither she, nor our children, will ever know how much I have cherished their encouragement through these times. I devote the future to them.

I would like to thank Mr. John Beeston for serving as my thesis director and a "neater, sweeter, slicker, quicker" role model. His realistic, keep-it-simple attitude has been a bright spot in my anesthesia education. I will never be able to repay the number of hours he has given.

I would also like to thank Mr. William Hartland for his part in this project as well as his student-centered approach to education. Bill has been a source of faith and support to me throughout my time at VCU. He has always taken time for me when I needed someone to lean on.

Lastly, I would like to thank Dr. Abey Albert for his efforts in this work. Abey has always shown genuine concern and support for students. He has supported my research efforts from the beginning, and asked for nothing in return.

Table of Contents

| | Page |
|--|------|
| List of Tables..... | v |
| List of Figures..... | vi |
| Abstract..... | vii |
| Chapter One: Introduction..... | 1 |
| Statement of Purpose..... | 4 |
| Statement of the Problem..... | 4 |
| Hypotheses..... | 4 |
| Variables..... | 5 |
| Independent..... | 5 |
| Dependent..... | 5 |
| Definition of Terms..... | 6 |
| Flow Rate..... | 6 |
| Delivered Temperature..... | 6 |
| Prewarmed Crystalloid Solution..... | 6 |
| Thermal Jacket™..... | 6 |
| Conventional Blood Warming Apparatus..... | 6 |
| Assumptions..... | 6 |
| Limitations..... | 7 |
| Conceptual Framework..... | 7 |
| Thermoregulation..... | 7 |
| Pathophysiology of Hypothermia..... | 9 |
| Hypothermia During Anesthesia..... | 12 |
| Prevention of Hypothermia..... | 15 |
| Effects of Blood Warming..... | 18 |
| Effects of Crystalloid Warming..... | 23 |
| Summary..... | 25 |
| Chapter Two: Review of Literature..... | 26 |
| Methods of Warming Blood and Crystalloids..... | 26 |
| Summary..... | 34 |

| | Page |
|--|------|
| Chapter Three: Methodology..... | 36 |
| Design..... | 36 |
| Population and Sample..... | 36 |
| Treatment Groups..... | 37 |
| Procedure..... | 38 |
| Instrumentation..... | 40 |
| Data Analysis..... | 42 |
| Chapter Four: Results..... | 43 |
| Group Effect..... | 46 |
| Flow Rate Effect..... | 46 |
| Chapter Five: Discussion..... | 48 |
| Hypothesis Testing..... | 48 |
| Correlation with Previous Studies..... | 49 |
| Flow Rate Effect..... | 52 |
| Group Effect..... | 52 |
| Limitations..... | 52 |
| Recommendation for Further Study..... | 53 |
| Conclusion..... | 54 |
| References..... | 55 |
| Vita..... | 60 |

List of Tables

| Table | Page |
|--|------|
| 1. Temperature of Fluids from Container to Distal Site by Group..... | 44 |
| 2. Temperature of Fluids from Container to Distal Site by Flow Rate..... | 45 |

List of Figures

| Figure | Page |
|---|------|
| 1. Thermal Jacket™ intravenous insulation device..... | 37 |
| 2. Infusion setup..... | 39 |

Abstract

EFFICIENCY OF THE THERMAL JACKET™ ON THE DELIVERED TEMPERATURE OF PREWARMED CRYSTALLOID INTRAVENOUS FLUID

Danny R. Bowen, BSN

School of Allied Health Professions--Virginia Commonwealth University, 1989.

Major Director: John T. Beeston, BS, CRNA

A quasi-experimental research design was used to determine the relationship between the flow rate and the delivered temperature of prewarmed crystalloid intravenous solutions when using the Thermal Jacket™, an insulation device designed for intravenous fluid bags, as compared to a conventional blood warming apparatus. One control and three experimental groups were used.

Fluids in Group 1 (control group), Group 2, and Group 4 were prewarmed in a microwave oven to 41.45 ± 1.05 °C. Fluids in Group 3 were left near ambient room temperature and measured at 22.05 ± 0.45 °C. Fluids in the control group were infused through a standard intravenous pump tubing, 270 cm in length, using no temperature maintenance device. Fluids in Group 2 and Group 3 were delivered through a standard intravenous pump tubing connected to a blood warming coil which was immersed in a water bath blood warmer. The distance from the exit point of the blood warmer to the distal end of the infusion line measured 174 cm. Fluids in Group 4 were placed in a Thermal Jacket™ and

delivered through standard intravenous pump tubing. After a baseline measurement, temperatures were recorded for all groups at two sites at 10 minute intervals over a 60 minute period. One site was the lower portion of the IV solution bag, and the other site was a point 2 cm from the distal end of the infusion set. Temperatures were measured at flow rates of 100, 250, 500, 750, and 1,000 ml/hr for each group.

Analysis of variance showed a highly significant group effect on the delivered temperature. A Bonferroni multiple comparisons test indicated no statistically significant difference between the delivered temperatures of Group 3 (room temperature fluid + blood warmer) and Group 4 (prewarmed fluid + Thermal Jacket™). Group 2 (prewarmed fluid + blood warmer) showed a significantly higher delivered temperature ($p < .05$) than the other groups, and the control group (prewarmed + no temperature maintenance device) showed a significantly lower delivered temperature ($p < .05$). Analysis of variance also showed a highly significant flow rate effect on delivered temperature. A Bonferroni multiple comparisons indicated a significant difference ($p < .05$) between the flow rates of 100, 250, 500, and 750 ml/hr, with the higher flow rates resulting in higher delivered temperatures. There was no significant difference noted between the delivered temperatures at 750 and 1,000 ml/hr.

The Thermal Jacket™, used with prewarmed intravenous fluids, was as effective as the conventional method of delivering warmed fluids. Also, within the range of flow rates studied, faster flow rates tended to yield a higher delivered temperature.

Chapter One

Introduction

Humans, as homeothermic animals, regulate body temperature by balancing heat production with heat loss. This balance is controlled by centers in the hypothalamus. The body responds appropriately, under normal circumstances, to temperatures above and below the set-point, or "normal" temperature range of 36.6-37.5 °C (Berne & Levy, 1983; Cabanac, 1975; Elder, 1984). When the body's core temperature rises above normal, it is considered to be hyperthermic; when it falls below normal, it is considered to be hypothermic. Hypothermia is further classified as mild (34-36.5 °C), moderate (28-33.5 °C), deep (17-27.5 °C), or profound (4-16.5 °C) (Elder, 1984).

Hypothermia affects all body systems to some degree. Moderate to profound hypothermia depresses cardiovascular function as evidenced by changes in the blood pressure, pulse, and electrocardiogram. It also depresses the respiratory system and decreases the oxygen delivery to the peripheral tissues. The central nervous system is depressed to the point of unconsciousness and renal function is decreased to a degree which hinders clearance of toxic substances and metabolic waste. Enzyme and hormone activity are decreased as are normal

clotting mechanisms. This degree of multisystem dysfunction is normally avoided by appropriate physiological responses to hypothermia.

There are several factors related to anesthesia and the operating room environment which could place every patient at risk for developing hypothermia. The first of these is that anesthetic agents themselves interfere with the body's normal response mechanisms to changes in temperature (Flacke, Flacke, Ryan, & Britt, 1983; Lonning, Skulberg, & Abyholm, 1986; Vale, 1973). Secondly, intravenous fluids and blood products are often infused near or below room temperature and contribute to hypothermia (Boyan & Howland, 1961, 1963; Norman, Ahmad, & Zeig, 1986). The third factor is that anesthetic gases are delivered cold and dry, leading to increased heat loss (Lilly, 1986; Yale, 1973). The fourth mechanism is that the modern operating room is typically maintained at an ambient temperature of 19-21 °C, which is well below body core temperature. This low temperature, coupled with the high air turnover, contributes significantly to the development of hypothermia (Holdcraft & Hall, 1978; Lilly, 1986; Morris & Wilkey, 1970). Lastly, cold irrigation fluids are often instilled in open body cavities and increase the heat loss during surgery (Goldberg & Roe, 1966; Holdcraft & Hall, 1978).

Because the operating room environment, surgical procedures, and anesthesia interventions potentially induce heat loss in many patients, the anesthetist must take certain measures to avoid the development of hypothermia, particularly in high risk patients. Anesthetists employ active and passive measures to prevent this heat loss. Passive means of preventing hypothermia

include increased ambient room temperature, liberal use of blankets and drapes, the use of a heat and moisture exchange unit in the anesthesia breathing circuit, and the use of warmed irrigation solutions (Elder, 1984; Goldberg, 1988; Haslam & Nielsen, 1986; Lilly, 1986; Morris, 1971; Morris & Wilkey, 1970; Roizen, L'Hommedieu, Wylie, & Ota, 1980; Shanks, 1975). Active means of preventing hypothermia include warming-blankets, heated and humidified inspiratory gases, warm gastrointestinal or peritoneal irrigation, radiant heat sources, and extracorporeal heat exchange units. One must remember, however that all of these active means have inherent risks, complications, and inconveniences (Conahan, Williams, Apfelbaum, & Lecky, 1985; Fried, Satiani, & Zeeb, 1986; Goudsouzian, Morris, & Ryan, 1973; Harnett, Pruitt, & Sias, 1983; Morris & Kumar, 1972; Papenburg, English, Foot, Farias, & Hinchey, 1987; Pickering, Bristow, & Craig, 1977; Scott, 1967; Shanks, 1975; Stone, Downs, Paul, & Perkins, 1981). Another popular active means of preventing hypothermia involves warming all intravenous infusions. This technique is effective when used alone or in combination with other methods (Boyan & Howland, 1961, 1962, 1963; Buckhold et al., 1978; Copping, Mather, & Winkler, 1972). Warming is accomplished by either the prewarming of fluids or the infusion of fluids through devices designed to warm them immediately before administration. These blood warming devices, though cumbersome, are effective within certain ranges of infusion rates (Baker, 1985; Cooter, 1987; Rymers, 1988).

Prewarming fluids is effective only as long as the heat can be retained by the fluid prior to infusion. A new device has been developed which potentially

increases the length of time that a bag of intravenous fluid retains its original prewarmed temperature without the use of bulky, cumbersome equipment. Its use with prewarmed fluids, therefore, may decrease both the incidence and severity of hypothermia in the operating room setting. This device, the Thermal Jacket™, is lightweight, easy to use, and potentially effective in infusing warmed fluids to the surgical patient.

Statement of Purpose

The purpose of this study is to determine the relationship between the flow rate and the delivered temperature of prewarmed crystalloid intravenous solutions when using the Thermal Jacket™ as compared to a conventional blood warming apparatus.

Statement of the Problem

Is there a difference between the Thermal Jacket™, used with prewarmed intravenous fluids, and conventional warming methods in maintaining the delivered temperature of crystalloid solutions at various flow rates in the operating room environment?

Hypotheses

This study addresses the following hypotheses:

1) there will be no difference between the delivered temperatures of prewarmed and room temperature fluids using a blood warmer apparatus;

2) there will be no difference between the delivered temperatures of room temperature fluids using a blood warmer apparatus and prewarmed fluids using no temperature maintenance device;

3) there will be no difference between the delivered temperatures of room temperature fluids using a blood warmer apparatus and prewarmed fluids using the Thermal Jacket™;

4) there will be no difference between the delivered temperatures of prewarmed fluids using a blood warmer apparatus when compared with no temperature maintenance device;

5) there will be no difference between the delivered temperatures of prewarmed fluids using the Thermal Jacket™ when compared with no temperature maintenance device; and

6) there will be no difference between the delivered temperatures of prewarmed fluids using the Thermal Jacket™ when compared with a blood warmer apparatus.

Variables

Independent. The independent variables were (a) the flow rate, and (b) the temperature maintenance device.

Dependent. The dependent variable was the delivered temperature.

Definition of Terms

Flow rate. Flow rate is the volume of fluid delivered via an infusion pump expressed in milliliters per hour (ml/hr). Flow rates in this study ranged from 100 ml/hr to 1,000 ml/hr.

Delivered temperature. Delivered temperature is the temperature of the fluid, expressed in degrees Celsius ($^{\circ}\text{C}$), at the distal end of the infusion tubing as measured by an inline thermistor.

Prewarmed crystalloid solution. Prewarmed crystalloid solution is a commercially prepared intravenous solution containing no oncologically active substance. Crystalloid solution in this study refers to lactated Ringer's solution in 1,000 milliliter (ml) bags warmed to approximately 42°C in a microwave oven.

Thermal Jacket™. The Thermal Jacket™ is an insulation device for intravenous fluid bags (Smith Medical Systems, Inc., Astoria, OR).

Conventional blood warming apparatus. A conventional blood warming apparatus consists of a heated, fluid-filled, metal heat sink, an associated fluid warming coil immersed in the heated fluid, and a length of extension tubing connecting the coil in the heat sink to the patient's intravenous catheter.

Assumptions

1. All infusion pumps will deliver the rate of flow at which they are set.
2. The thermometers will provide accurate readings of the temperature of the fluids.

Limitations

1. Five thermometers were used in this study.
2. Four infusion pumps were used in this study.
3. Room temperature was set using a wall thermostat and measured, but not controlled.

Conceptual Framework

Thermoregulation. Homeothermic animals, such as humans, regulate their body temperatures by balancing heat production with heat loss. This balance involves a complex set of responses which have been only partially delineated. It is known that mechanisms such as autonomic and behavioral responses play a role in thermoregulation. It is also known that there are three components to thermoregulation. These components are the afferent temperature sensors, the efferent motor systems, and the control system.

The mechanisms involved in thermoregulation can be described in terms of their responses to both increased and decreased body temperatures. The autonomic response to an increased temperature is stimulation of sweating and dilation of cutaneous blood vessels. Sweating increases heat loss by evaporation; and cutaneous vasodilation increases heat loss by radiation. Hormonal changes to increased temperature include a decrease in thyroxine release by the thyroid gland. This decrease reduces metabolism, which in turn decreases heat production. Behavioral responses to increased temperature include change of posture and avoidance of hostile temperatures. The change of posture serves to

increase the surface to mass ratio, and thereby increases the heat exchange between the animal and the environment.

The autonomic response to a decreased temperature is piloerection and constriction of cutaneous blood vessels. Piloerection forms a layer of air dead space around the skin which decreases evaporative losses; and cutaneous vasoconstriction reduces radiant heat loss. The hormonal response to a decreased temperature is an increased output by the thyroid gland which increases metabolism and thereby increases heat production. Behavioral responses to decreased temperatures include a change of posture and avoidance of hostile temperatures. The change of posture with decreased temperatures serves to decrease the surface to mass ratio and decrease the heat exchange between the animal and the environment (Berne & Levy, 1983; Flacke et al., 1983; Cabanac, 1975).

As previously stated, there are three components to thermoregulation. Two of these, the afferent temperature sensors and the efferent motor systems, are regulated by the third, the control system. The afferent temperature sensors are divided into the peripheral sensors and the central sensors. It is estimated that the central sensors are three times more predominant than the peripheral sensors in humans (Cabanac, 1975). The most efficient responses to temperature changes, however, have been found to occur when both central and peripheral systems are intact (Downey, Miller, & Darling, 1969). The central sensors are located in the anterior hypothalamus and the spinal cord. The peripheral sensors are located nonuniformly in skin, mucosa, and some deep tissues. It has been

postulated that the uneven distribution of peripheral sensors suggests that weighted integrated input from the peripheral sensors exists which determines the sensitivity of the central regulatory system (Flacke et al., 1983). This peripheral regulation of the central sensors may account for the observation that the most efficient responses occur when both systems are intact. The efferent motor systems include heat generating mechanisms such as skeletal muscle activity and heat dissipating mechanisms such as vasodilation. Although it is not the only contributor, the hypothalamus seems to be the center of thermoregulation (Berne & Levy, 1983; Cabanac, 1975). In addition to sensing temperature directly, it also receives input from the afferent system and regulates the efferent system to maintain a relatively constant temperature of 36.6-37.5 °C. This set point temperature is the critical temperature above which heat dissipation is activated and below which heat generation and conservation are stimulated.

Pathophysiology of hypothermia. Hypothermia has a number of detrimental effects on the normal physiology of the body. The cardiovascular, respiratory, nervous, renal, endocrine, and musculoskeletal systems are all adversely affected by hypothermia. These detrimental effects are a function of both the duration and the degree of hypothermia (Elder, 1984).

The cardiovascular system is one of the major systems affected by hypothermia. With core temperatures ranging from 33-35 °C, there is an increase in heart rate, central blood volume, and mean arterial blood pressure. Cardiac output can increase up to 500% due to peripheral vasoconstriction resulting from an increased level of catecholamines. At core temperatures of 30-

33 °C, there is a reflex decrease in heart rate, cardiac output, and blood pressure (Elder, 1984; Lonning et al., 1986). As the core temperature drops below 30 °C, there is a propensity toward dysrhythmias. The electrocardiogram (ECG) demonstrates these dysrhythmias, as well as other characteristic electrical changes with an increasing magnitude of hypothermia. There are typically no changes, except in rate, noted on the ECG, at temperatures above 33 °C. Below this core temperature there are increases in the P-Q and Q-T intervals, widening of the QRS complex, bradycardia, and development of a J-wave, all associated with a slowed myocardial conduction. There is also an increased likelihood of developing atrial fibrillation, atrial flutter, nodal rhythm, or second degree heart block. Below a core temperature of 30 °C there is progressive widening of the QRS complex and extreme bradycardia with frequent occurrence of ventricular extrasystoles. Atrial fibrillation typically occurs due to atrial distention, and as the temperature continues to decrease, there is an increased incidence of more fatal dysrhythmias. Below 27-29 °C there is a high incidence of ventricular fibrillation progressing to asystole at temperatures below 20 °C (Churchill-Davidson, 1955; Elder, 1984; Little, 1959; Lonning et al., 1986).

The respiratory system also undergoes extreme changes in response to hypothermia. The initial response to core temperatures below 35 °C is an increased respiratory rate corresponding to an increased oxygen demand caused by shivering. As the temperature falls below 33 °C, there is a steady decrease in the respiratory rate, approaching 1-2 gasps per minute at temperatures below 30 °C. At these very low temperatures, there is an increased production of

bronchial secretions with an increased incidence of bronchospasm. Hypothermia shifts the oxyhemoglobin dissociation curve to the left, decreasing the release of oxygen from the hemoglobin molecule to the tissues. This effect on the oxyhemoglobin dissociation curve is offset, somewhat, by a shift to the right caused by metabolic acidosis. Metabolic acidosis accompanies the peripheral vasoconstriction seen with hypothermia. Functionally, then, the oxyhemoglobin dissociation curve changes have minimal consequences (Elder, 1984; Lilly, 1986; Little, 1959; Lonning et al., 1986).

Other physiological systems show fewer, though no less profound, changes with hypothermia. The central nervous system shows mental impairment below 33 °C, progressing to a loss of consciousness below 30 °C, and an absence of activity on the electroencephalogram (EEG) below 20 °C (Elder, 1984; Little, 1959; Lonning et al., 1986). The renal system demonstrates a "cold diuresis" between 33 °C and 35 °C due to the peripheral vasoconstriction associated with hypothermia. As the core temperature drops below 33 °C, there is a decreased glomerular filtration rate which decreases the kidneys' ability to clear drugs and their metabolites (Elder, 1984; Lonning et al., 1986). With hypothermia comes hyperglycemia and ketosis due to a decreased release of insulin from the pancreas and a decreased insulin effect at the peripheral binding sites. There is also a decrease in overall enzyme activity, with the brain being more sensitive than other organs (Elder, 1984; Lonning et al., 1986). The musculoskeletal system responds to hypothermia with vasoconstriction and shivering. Below 33 °C there is decreased shivering and a general stiffness of muscles and joints due to

muscular dysfunction as a result of impaired conduction in the peripheral nerves (Little, 1959; Lonning et al., 1986). There is a progressive prolongation of the clotting time, along with other coagulation defects, accompanying increased levels of hypothermia (Churchill-Davidson, 1955; Lonning et al., 1986).

It is evident that as the degree of hypothermia increases, the degree of dysfunction in most systems of the body increases. Though some of these may be beneficial, the overall effect of hypothermia is a deleterious one which requires countermeasures to avoid eventual death.

Hypothermia during anesthesia. Hypothermia during anesthesia and surgery is common. Two factors which contribute to intraoperative hypothermia are the nature of the operating theater environment and the effects of anesthesia on thermoregulation. There are four mechanisms for this heat loss in the anesthetized patient: (a) conduction, (b) convection, (c) radiation, and (d) evaporation. Conduction is the transfer of heat from the body to colder surfaces which it contacts. Convection is the heat loss to cooler, surrounding air currents and can be thought of as the wind chill factor. Radiation is the heat loss that occurs between objects of different temperatures not in contact with each other. The body radiates heat from uncovered areas, especially the scalp and upper torso. Evaporation is the loss that occurs when the skin and mucus membranes provide heat to transform liquid water to a gaseous state.

The first factor contributing to intraoperative hypothermia is the operating room environment. The typical operating suite is conducive to heat loss leading to hypothermia. The ambient room temperature is commonly maintained at 19-

21 °C, primarily for the comfort of the surgical team. At those temperatures, without any measures taken to avoid heat loss, most patients will become hypothermic (Morris & Wilkey, 1970). Even at temperatures as high as 24 °C, many patients still become hypothermic (Holdcraft & Hall, 1978; Lilly, 1986). Much of the heat loss can be accounted for solely by the ambient temperature. This is demonstrated by the greatest amount of heat loss occurring in the first hour in the operating room and in the time between skin closure and delivery to the recovery room.

There are, however, factors other than room temperature that contribute to hypothermia. Modern operating rooms have a high air turnover which increases the likelihood of convective heat loss. The operating table, unless heated, will be near room temperature, and this increases the conductive heat loss. Irrigation fluids are frequently used at room temperature and this increases both conductive and convective heat loss. Another common factor is the increased surface area available for conductive, convective, and radiative heat losses when the integrity of the skin is interrupted by the surgeon's knife. All of these make the modern operating suite an ideal location for induction of unintentional hypothermia.

The second factor contributing to intraoperative hypothermia is the effect of anesthetic agents on thermoregulation. While the operating room environment disrupts the balance of thermoregulation, anesthesia tips the scales in favor of hypothermia. Many of the anesthetic agents administered impair the compensatory mechanisms normally used by the body in thermoregulation. Many anesthetics cause cutaneous vasodilation, reduce thermogenic shivering, lower the

basal metabolic rate decreasing heat production, or act directly to impair the temperature regulation center in the hypothalamus (Flacke et al., 1983; Lonning et al., 1986; Yale, 1973).

Even the intravenous fluids administered increase the risk of developing hypothermia. Intravenous fluids are usually infused at room temperature which may be almost 20 °C lower than the core temperature of the body. Blood is typically infused at even lower temperatures. Blood is stored at 4-6 °C and, other than incidental warming, is infused near that frigid temperature. Cold, dry anesthetic gases are administered to the patient which cause evaporative and convective heat loss (Lilly, 1986; Yale, 1973). The combination of anesthesia and operating theater environment set the scenario for a hypothermic patient, especially if that patient is already at risk for development of hypothermia.

There are a number of patient characteristics which have been found to be related to an increased incidence of hypothermia. People at either extreme of age, namely young children and elderly adults, tend to have a higher incidence of hypothermia during anesthesia. The explanation for this, as well as an increased incidence of hypothermia in cachectic patients, seems to be related to decreased muscle mass, less vigorous shivering, and a resultant decrease in heat production. Patients with massive injuries, exhaustion, and undernourishment also have a decreased ability to produce heat; therefore, they have an increased risk of developing hypothermia (Lilly, 1986; Lonning et al., 1986; Roe, Goldberg, Blair, & Kinney, 1966).

There are also a number of characteristics associated with the surgery and surgical requirements that increase the incidence of hypothermia. Any procedure requiring the opening of any major body cavity increases the risk of hypothermia as does any procedure which necessitates infusion of large volumes of fluid or blood. (Goldberg & Roe, 1966; Lilly, 1986). As stated before, ambient temperature plays a major role in hypothermia, so any surgical condition which requires low ambient temperatures will put the patient at risk for heat loss.

Patients who have substantial heat loss during surgery must regain this heat in the postoperative period. Unfortunately, those patients at increased risk for development of hypothermia are also those least capable of recovering the lost heat by increasing heat production in the postoperative period. For example, elderly patients have been found to require twice as much time to return to their preoperative temperatures when compared to younger adults, even though their immediate postoperative temperatures may be similar (Vaughn et al., 1981).

Prevention of hypothermia. Since hypothermia in the operating room is quite common, a number of measures have been utilized to prevent or counter heat loss. These methods can be divided into two broad categories: passive and active warming. In the first of these, passive warming, the patient's endogenous heat production is the source of thermal energy and is augmented by decreasing the heat loss. Passive measures are typically easy to apply and have minimal risks associated with them. One passive method is to increase the ambient temperature at least until the patient is prepped and draped. At room temperatures above 24 °C very few patients will become hypothermic, whereas, at

temperatures below 21 °C almost all patients become hypothermic (Morris, 1971; Morris & Wilkey, 1970; Roizen et al., 1980). Another passive method is covering as much of the patient as is practical with some type of insulating material. This material can be towels, cotton blankets, mylar-aluminum "space blankets", or even aluminum foil or plastic wrap. The most important areas to cover are the head, neck, and shoulders because these areas maintain perfusion even when the core temperature drops (Elder, 1984; Lilly, 1986; Shanks, 1975). A third passive method of decreasing heat loss is the use of a heat-moisture exchange unit ("artificial nose") inserted between the anesthesia breathing circuit and the endotracheal tube (Goldberg, et al., 1988; Haslam & Nielsen, 1986). This decreases the energy required to warm and humidify the cold, dry gases administered to the patient (Lilly, 1986). The fourth passive method is the use of warmed irrigation solutions during surgery. These passive methods are relatively easy to employ, cost effective, and generally risk free.

The second category of preventing or countering heat loss is active warming. With these measures, external energy is supplied to supplement the patient's own heat production. Active methods are usually more involved and potentially more dangerous than passive means. For example, the seemingly innocuous use of a warming-blanket can be hazardous. With warming blanket temperatures of 40-42 °C serious thermal injuries have been reported (Scott, 1967).

Use of warming blankets is widespread in spite of studies which have shown them to be potentially dangerous as well as ineffective in preventing or reversing hypothermia in patients weighing more than 10 kg (Goudsouzian et al., 1973;

Morris & Kumar, 1972; Stone et al., 1981). Another active method, heating and humidifying of inspiratory gases, has been shown to be effective in preventing and even in reversing hypothermia (Conahan et al., 1985; Shanks, 1975; Stone et al., 1981). This method, however, can cause thermal damage to the respiratory mucosa as well as potential overhydration due to the volume of fluid administered in the humidified gases via the breathing circuit (Harnett et al., 1983).

Gastrointestinal warming is another active method used to prevent or counter hypothermia. With this method, gastric lavage is accomplished with warm irrigation fluid. Gastrointestinal warming has the advantage of applying heat directly to the core of the body without penetrating any tissues. The only risk involved with this method is mechanical trauma from the insertion of the irrigation tube.

A fourth method is the use of a radiant heat source. This is acceptable in the recovery room, but, because of the request from the surgical team for cooler ambient temperatures; this method is not practical except for infants (Flacke et al., 1983; Lilly, 1986).

The fifth and sixth methods are peritoneal irrigation with warmed fluid and warming the patient's blood using an extracorporeal heat exchanger. These two methods have been used to reverse profound hypothermia, and therefore, could be used to attenuate the heat loss during surgery, but their use is neither practical nor risk-free. Both methods are fast and both can include dialysis of toxic substances from the blood. These advantages make their use more suited for an

emergency department than an operating room. Both have many inherent risks, and are, therefore, not useful in routine operating room circumstances (Fried et al., 1986; Harnett et al., 1983; Papenburg et al., 1987; Pickering et al., 1977).

The final active method of preventing hypothermia concerns the warming of all fluids and blood products infused during surgery. This has been found to prevent and even reverse hypothermia, even in patients receiving 18 or more liters of fluid and blood over a few hours (Boyan & Howland, 1961, 1962, 1963; Buckhold et al., 1978; Copping et al., 1972).

Effects of Blood Warming

The deleterious effects of cold blood infusion were seemingly not appreciated until the mid 1950s. In 1955, Howland, Schweizer, Boyan, and Dotto published a study concerning the physiologic alterations resulting from massive blood replacement. This study looked at two groups of patients who received multiple transfusions during the course of surgery.

In group one ($n = 130$), each patient received 10 or more units of citrated blood, and in group two ($n = 123$), each patient received 5-9 units of citrated whole blood. In group one, there was a 43% incidence of one of two major complications. The first complication concerned a defect in the clotting mechanism. This defect appeared to be directly related to the volume of blood administered. The second major complication involved the cessation of cardiac function in 22 patients, all of whom received more than 10 units of blood. In the 11 patients monitored by electrocardiography, these cardiac dysfunctions were

identified as either ventricular fibrillation or asystole. The incidence of these dysfunctions appeared to be correlated not with the volume of blood administered, but with the speed of blood administration. It was concluded, "the exact mechanism by which rapid transfusion of large quantities of citrated blood produces either cardiac standstill or ventricular fibrillation is still in doubt ..." (Howland, Schweizer, Boyan, & Dotto, 1955). Hyperkalemia or citrate intoxication with resultant hypocalcemia was thought to play a role in the cardiac dysfunction.

Interestingly, the authors stated "marked cooling of the viscera is a fact observed by many surgeons after massive blood replacement and is usually premonitory of impending disaster" (Howland et al., 1955). The authors also noted that a direct correlation between the degree of cooling and the onset of ventricular fibrillation appeared to exist. Cooling, however, was not listed as a possible major factor in the complications studied.

In 1956, three of the same authors, Howland, Boyan, and Schweizer, examined ventricular fibrillation during massive blood replacement. In this study of 253 patients who received 2,500 ml or more of citrated banked blood, nine instances of ventricular fibrillation were noted. Six of these instances of ventricular fibrillation occurred in patients who had received more than 5,000 ml. The most consistent factor in the development of ventricular fibrillation was found to be rapid, intravenous, citrated blood replacement. It was concluded, however, that this rapid rate of administration was not the only factor responsible.

Other possible factors contributing to cardiac dysrhythmias included hyperkalemia, citrate intoxication, pH of the blood, and, more importantly, hypothermia. The authors proposed that infusion of cold, banked blood resulted in a stream of cold blood being delivered directly to the right side of the heart and concluded "it is possible that this cooling of the myocardium may lead to cardiac arrhythmias and ventricular fibrillation" (Howland, Boyan, & Schweizer, 1956). Also noted were classic alterations in the electrocardiogram which have since been associated with hypothermia such as peaked, elevated T waves, prolonged Q-T interval, and wide, bizarre QRS complexes. In conclusion, the authors speculated that lowered myocardial temperature may be one of the etiologic factors in the development of ventricular fibrillation.

In 1961, Boyan and Howland examined the relationship between the temperature of administered blood, the patient's esophageal temperature, and physiological response. In this study, a thermocouple was inserted into the esophagus of patients to a point behind the right atrium to attain approximate cardiac temperatures. Two patients received 6,350 ml and 21,000 ml, respectively, of cold banked blood at 6,600-9,000 ml/hr and demonstrated progressive cardiac dysfunction. The first signs of cardiac change were noted at 29-33 °C. These signs consisted of prolongation of the ST interval, bradycardia, ventricular extrasystoles, and hypotension. At 29.8-32 °C all cardiac function ceased.

Boyan and Howland (1961) reported the first clinical use of a blood warming apparatus. Three patients received 7,800-9,600 ml of 33-35 °C blood through a blood warmer at a rate of 3,600 ml/hr. Temperatures for the three

patients remained above 35.7 °C and none exhibited signs of cardiac dysfunction. The authors concluded that cardiac difficulties associated with massive blood replacement could be related to the degree of hypothermia of the heart.

All of the studies reviewed up to this point have been anecdotal or descriptive and have not provided statistical analyses nor specific methodologies. These researchers merely identified the occurrence of hypothermia as a result of cold, banked blood administration without utilizing true or quasi-experimental methodologies to validate their findings.

In 1963, Boyan and Howland reported a retrospective, nonrandomized study of 81 patients who received 3,000 ml or more of blood, cold or warm, at rates of at least 3,000 ml/hr. Of the 36 patients who received cold blood, 25 had infusion rates of 3,000-6,000 ml/hr and 12 of these experienced cardiac arrest in the operating room. The remaining 11 patients received cold blood at greater than 6,000 ml/hr and 9 experienced cardiac arrest in the operating room. There were 45 patients who received blood via the warming device. Of these, 40 received blood at 3,000-6,000 ml/hr and none of these experienced cardiac arrests. The remaining five patients received warmed blood at rates exceeding 6,000 ml/hr and only one patient experienced a cardiac arrest in the operating room.

The authors found a statistically significant ($\chi^2 = 29.0$, $p < .01$) higher incidence of cardiac arrest in those patients receiving cold blood when compared to those receiving warm blood. A statistically significant ($\chi^2 = 20.4$, $p < .01$) higher incidence of cardiac arrest was also found in patients receiving blood, warm or cold, at rates greater than 6,000 ml/hr as compared to those receiving

blood at rates of 3,000-6,000 ml/hr. The authors attributed part of these differences in the cardiac arrest incidence to the patient's condition since the patients who required rapid administration were most likely experiencing problems not controlled for in this retrospective study.

In 1964, Boyan reported on an additional number of patients who received blood via the blood warming device and statistically reexamined his 1963 study to include these patients. This study, as the first, was retrospective and nonrandomized. The same cold blood infusion group was used as before and more warm blood infusion patients were added to bring the total to 118 patients who received blood via the blood warming device. Once again a statistically significant difference ($\chi^2 = 44.5$, $p < .01$) was found between the warm and cold groups with the patients receiving cold blood demonstrating a higher incidence of cardiac arrest in the operating room. A statistically significant difference ($\chi^2 = 35.7$, $p < .01$) was also found between the patients receiving blood at 3,000-6,000 ml/hr as compared to those receiving blood at greater than 6,000 ml/hr. As before, the faster infusion group demonstrated a higher incidence of cardiac arrest. All patients included since the previous study received blood via the blood warmer. No additional patients were infused cold blood. The author failed to discuss any changes in the patient population, criteria used for blood administration, surgical technique changes, or other factors that may have accounted for decreased morbidity and mortality.

In 1986, Fried et al. reported on a rapid solution administration set (RSAS). This study described the in vivo use of the RSAS on one mongrel dog. The dog

was anesthetized, exsanguinated of 1,200 ml of blood, and then administered the 1,200 ml of autologous blood via the RSAS at 43,800 ml/hr. After 74 minutes, the dog was again exsanguinated of 1,200 ml of blood and then administered 1,200 ml of 4 day old homologous blood via the RSAS at 42,600 ml/hr. Hemodynamic measurements, a battery of laboratory tests, and vital signs were recorded throughout the study period.

Hemodynamic parameters, laboratory values, and vital signs were reported in a table without statistical analyses. On visual inspection, it appeared that post-infusion values of all parameters measured were essentially the same as baseline measurements. The dog's temperature never fell more than 1.2 °C below its baseline temperature. The authors then concluded that this device is probably effective in maintaining normothermia in a trauma patient. This appeared to be a rather strong generalized conclusion from a study that (a) involved one mongrel dog, (b) made no statistical analysis, (c) involved no control group, and (d) made no mention of the setting where the study took place or to any control of other variables. For example, there was no mention of the ambient room temperature. Many studies have found ambient room temperature to be a significant contributor to maintenance of normothermia in surgical patients.

Effects of Crystalloid Warming

Newman (1971) reported a nonrandomized, controlled study of 33 patients receiving one of three interventions to prevent the development of hypothermia during surgery. Patients in Group I (Control group) were operated on without

any attempt to control their temperatures. Group II patients were placed on a warming blanket (40-42 °C) throughout the operation, and Group III patients were placed on a warming blanket and given warmed (37 °C) intravenous fluids and blood. Esophageal temperatures were measured every 15 minutes after induction of anesthesia.

Fourteen of the 15 patients in Group I had a 2.0-4.6 °C decline in esophageal temperature. The remaining Group I patient had a 0.8 °C fall in temperature throughout the surgery. Seven of the eight Group II patients demonstrated a 0.5-2.0 °C decline in temperature during the surgery while the remaining patient had a 0.2 °C increase in temperature. The 10 patients in Group III ranged from a 0.5 °C drop in esophageal temperature to a 1.3 °C gain in temperature. Visual examination of the data indicated that the warming blanket alone decreased the heat lost during surgery, and when combined with infusion of warm intravenous fluids, made the heat loss negligible. No definite conclusions can be reached, however, since no statistical analysis was reported.

Copping et al. (1972) reported a nonrandomized, controlled study of 16 mongrel dogs that were placed in controlled, hemorrhagic shock and subsequently volume repleted with lactated Ringer's solution at various temperatures. Group I (\underline{n} = 5) received infusions of fluids cooled to 4.5 °C. Group II (\underline{n} = 6) served as a control group and received infusions of fluids at a room temperature of 21.0-26.7 °C. Group III (\underline{n} = 5) received infusions of fluids warmed to 37.0 °C and infused at 33.3-34.4 °C. All solutions were infused at 3,000 ml/hr and all groups received similar volumes of intravenous fluid. Results of mortality rates,

esophageal temperatures, pulse changes, and ECG changes were presented, but analysis of data was confined to descriptive statistics. It appeared that the degree of hypothermia, cardiovascular changes, and mortality all increased as the infusion temperature decreased. While the study seemed to have controlled extraneous variables and demonstrated differences, it failed to provide statistical analyses to support any findings.

Summary

As we have seen, the detrimental effects of rapid, cold blood administration has been recognized since the late 1950s. Since that time, various methods of warming blood have been investigated. Researchers then studied the effects of administering warmed intravenous fluids. From this line of research, it would appear that the use of various blood warming devices for warming both blood and crystalloid fluid plays a major role in decreasing both morbidity and mortality resulting from hypothermia. One new concept in fluid warming is the Thermal Jacket™. Until now, no formal research has been done concerning its effectiveness in delivering warmed fluid.

Chapter Two

Review of Literature

Methods of Warming Blood and Crystalloids

A number of methods have been utilized in delivering warmed fluids and blood to the surgical patient. The first of these was first developed and reported in 1961 by Boyan and Howland. This blood warmer consisted of 24 feet of 4.5 mm diameter plastic tubing wrapped around a wire frame and immersed in a 20 liter water bath. The water in the bath was maintained near 37 °C by adding hot or cold water and was monitored using a bath thermometer. The one safety feature, a thermocouple, activated a warning light if the temperature in the bath exceeded 37 °C. Though bulky and cumbersome to use, this first blood warmer was an improvement over cold blood administration. At flow rates of 3,000 ml/hr this device warmed blood from a storage temperature of 4-6 °C to 35 °C. The warmer was less efficient at flow rates of 6,000 ml/hr, warming the cold blood to 33 °C. At higher flow rates of 7,200-9,000 ml/hr, the device was only able to warm the blood to 30.6-32 °C.

Baker (1985) investigated the effect of flow rate on heat loss in packed red blood cells warmed using a modern blood warmer (DUPACO,

Hemokinetitherm®) with a bath temperature maintained at 33-34 °C. In an operating room with an uncontrolled ambient temperature measured at 16.2-18.7 °C, flow rates of the blood were manipulated over a range of 50-4,000 ml/hr. Temperatures were measured at both the inlet and outlet ports of the blood warmer and again at the distal end of the infusion set. The blood used was initially near room temperature rather than at a typical storage temperature of 4 °C.

Baker (1985) found that at flow rates below 500 ml/hr, the delivered temperature of the blood was lower than the temperature at the inlet port of the blood warming unit. It was proposed that this heat loss may have been due to the colder temperature of the air near the floor, where the blood warmer was placed, and its effect on convective heat loss of the system. The use of the blood warmer at these flow rates may be counterproductive to the desired effect of delivering warmed fluids to the patient. At flow rates of 500-3,000 ml/hr there was a nonlinear, positive correlation between the flow rate and the delivered temperature of the blood indicating an ability to overcome convective heat losses that the slower flow rates experienced. Within this range, the faster flow rates resulted in higher delivered temperatures. At flow rates exceeding 3,000 ml/hr there was a steady decline in the delivered temperature. These flow rates, it was suggested, may have exceeded the heat transfer capabilities of the system used. Based on these findings, it was concluded that at flow rates between 500-3,000 ml/hr the system was effective in increasing the delivered temperature of room

temperature, banked blood. It was also concluded that outside of this flow rate range the system became ineffective and even counterproductive.

Cooter (1987) studied the Fenwal Dry Heat® blood warmer using a methodology similar to Baker's (1985), but with the following improvements: (a) the storage temperature of the blood was maintained at 1.2-4.3 °C before infusion; and (b) the blood warming device used was mounted on a pole next to the patient potentially, eliminating the high convective loss near the floor of the operating room. Flow rates of 100-8,000 ml/hr were used and were compared to the delivered temperature. Additional infusion pumps were added to achieve higher flow rates. Uncontrolled room temperature was measured at 19.8-20.8 °C.

It was found that the delivered temperature of the blood rose continually from flow rates of 100 ml/hr to 1,000 ml/hr. There was a decline in the delivered temperature from flow rates of 1,100 ml/hr up to 1,900 ml/hr, and then another steady increase in delivered temperature at flow rates of 2,000-7,992 ml/hr. Cooter concluded that the Fenwal Dry Heat® blood warmer became more effective as flow rates increased, and was more effective at high flow rates than the blood warmer used by Baker (1985).

What Cooter (1987) did not address was the presence of variations in the expected delivered temperatures at several flow rates. For example, decline in delivered temperature from 1,100-2,000 ml/hr was noted, but not the peaks representing maximum differences from predicted temperatures at about 1,000, 2,000, 3,000, 4,000, 5,000, 6,000, 7,000, and 8,000 ml/hr. On inspection, it appeared that these maximum differences occurred at flow rates corresponding to

the maximum output of these pumps or at points in the study when new pumps had to be added to the system. The reason for addition of new pumps is that each Travenol volumetric infusion pump used was only capable of administering fluids up to 999 ml/hr. As the experimental flow rate increased, it became necessary to connect additional pumps to accommodate this increase.

Rymers (1988) studied a DUPACO, Hemokinetitherm® blood warmer similar to that investigated by Baker (1985). This study examined the effect of flow rate and intravenous tubing length on the delivered temperature of warmed packed, red blood cells. The heat loss at low flow rates in Baker's (1985) study was attributed to conductive losses caused by contact between the infusion tubing and cold surfaces in the study environment. Improving on the methodology used by Baker, Rymers ensured that no part of the infusion tubing came in contact with any non-insulated object. With this modification, delivered temperatures were higher at all flow rates than temperatures at the inlet port of the blood warmer. The delivered temperature increased from flow rates of 100 ml/hr to 3,100 ml/hr at which time a steady state was reached with similar delivered temperatures being attained up to 5,700 ml/hr. Above this flow rate, a decline in delivered temperature was noted. It was also found that the greater the length of the infusion tubing, the more heat was lost between the blood warmer and the distal end of the infusion set. As with Cooter's (1987) study, Rymers' data showed fluctuations in the curve representing the relationship between flow rate and temperature at flow rates near the points where additional infusion pumps

had to be added. These fluctuations and relationships were not addressed in the discussion.

Zorko and Polsky (1986) investigated yet another method of delivering warmed blood to the surgical patient. This study was designed to examine the flow rates of blood delivered at various temperatures and through various circuits. The delivered temperature of blood, stored at 4 °C and divided into four research groups, was compared. In the control group (I), blood was infused through a blood administration set using no warming method. In a second group (II), stored blood was warmed using a Travenol blood warming coil and Hemokinetitherm® blood warmer. A third group (III) used the same infusion tubing as the control group, but in this group, 250 ml of 45 °C normal saline was added to the packed, red blood cells just prior to infusion. The fourth group (IV) combined both experimental methods (II & III) by adding warmed saline to the blood and infusing it through the blood warming device.

It was pointed out that blood used in the control group was warmed to a mean temperature of 18 °C as it traversed the length of the blood administration set. The addition of warmed saline to the blood resulted in a mean delivered temperature of 26 °C. Infusion of cold blood through a blood warmer yielded a mean temperature of 29 °C. The combination technique of adding warmed saline to the blood and infusing it through the warming device yielded the highest mean delivered temperature of 35.3 °C.

A number of possible problems exist in the methodology and conclusions. First, the units of blood in the control group (I) and in group II, which used the

blood warmer alone, were undiluted. Adding room temperature fluid to these groups, as was done with groups III and IV, would possibly have increased the initial blood temperatures above the stored temperature of 4 °C. This would have, therefore, decreased the difference in delivered temperatures between the diluted and undiluted groups using the same method of delivery. Second, the control group gained 14 °C without any intervention. An additional group, infusing unwarmed blood through a blood warming coil, but not using a blood warmer, may have determined how much of the temperature gain in the blood warming circuit was attributable to movement through the tubing exposed to ambient room temperature. A final major problem area with this study was the lack of flow rate control. Controlling the flow rate would have controlled the length of time the blood was exposed to ambient room temperature, and therefore determined the effect of room temperature exposure on delivered temperatures. Though the study was designed to examine flow rates, controlling the flow rates may have made the delivered temperature data more meaningful.

Aldrete (1985) compared the delivered temperatures of lactated Ringer's solution infused at 6,000 ml/hr using one of three methods of warming the fluid. In Group 1, fluid was passed through a Goldman-Rupp blood warmer set at 36 °C using a 180 cm intravenous tubing connected to the standard blood warming apparatus for a total length of 375 cm. Fluids in Group 2 were warmed in a Sears Kenmore™ microwave oven to 41.5 °C and delivered using only 180 cm of tubing. Group 3 used a combination of these two methods. Group 4, the control group, consisted of room temperature fluid delivered through the 180 cm

tubing using no warming methods. The control group maintained an approximate temperature of 18 °C both in the bag and at the distal infusion site. The unwarmed group using the blood warmer resulted in temperatures of 27 °C, while the combination method (microwave warming + blood warmer) increased the delivered temperature to 30 °C. The microwave warming alone, however, produced the highest delivered temperature of 33-34 °C.

Aldrete (1985) also examined the temperature of fluids in the bag and delivered temperature of fluids at two flow rates. It was found that fluid in the bag and fluid delivered to the distal site both cooled more per volume infused when the infusion rate was 1,200 ml/hr as compared to an infusion rate of 6,000 ml/hr. A possible problem with this finding is that the rate of cooling was not expressed in terms of time. It would appear logical that the length of time fluid is exposed to ambient room temperature could significantly effect the amount of heat lost in the fluid. Another possible shortcoming of this study was the lack of recorded specific temperatures. Very few temperatures were reported as exact values. Instead, most data was displayed in graphic form and showed only temperature trends. Though trends are informative, specific data could provide for statistical analysis.

Another method of fluid warming was investigated by Fried et al. (1986). This study examined the efficiency of an extracorporeal heat exchanger when used as part of a rapid solution administration set. This set was designed to administer large amounts of fluid quickly through a 8.5 French catheter. With a countercurrent waterflow of 20 liters per minute at a temperature of 40 °C, blood

was warmed from a storage temperature of 4 °C to a delivered temperature of 38-39 °C. There was a detailed explanation of the equipment and materials used to develop this administration set and a clear reporting of the findings. This study, however, used no control group and reported no statistical analysis of the data.

Norman et al. (1986) studied the effects of flow rate, time, and insulation on the delivered temperature of lactated Ringer's solution prewarmed in a warming cabinet. It was found that the higher the initial temperature of the solution, the higher the delivered temperature at all time intervals. Fluids infused at 1,000 ml/hr with an initial temperature of 44.0 °C had delivered temperatures of 33.9 °C at 5 minutes and 28.0 °C at one hour after removal from the warming cabinet. At the same rate, fluids with an initial temperature of 49.6 °C had delivered temperatures of 37.2 °C at 5 minutes and 31.0 °C at one hour. Fluids prewarmed to 50.0 °C and insulated using foam rubber as an insulating material had a delivered temperature of 37.0 °C which was approximately the same as the uninsulated fluids. However, at one hour, the insulated fluid had a higher delivered temperature of 33.8 °C. The control group maintained temperatures within 1 °C of ambient room temperature at all times measured.

It was concluded that at a slower rate of 500 ml/hr, fluids had to be prewarmed to higher temperatures to have delivered temperatures approximating the higher infusion rate groups. Fluids prewarmed to 58.8 °C had delivered temperatures of 36.0 °C at 5 minutes and 30.1 °C at 1 hour. With an initial temperature of 55.0 °C, the slower rate resulted in a delivered temperature of

28.9 °C at 1 hour. As a result, it was demonstrated that insulation of prewarmed bags of intravenous fluid helped maintain higher delivered temperatures throughout the one hour trial. It was also found that higher initial temperatures and higher flow rates resulted in higher delivered temperatures at all times studied.

Summary

The literature appears clear on the fact that administering cold intravenous fluid and blood can induce hypothermia in surgical patients. It also appears that delivering warm fluids and blood can decrease this incidence of hypothermia with its associated morbidity and mortality. A number of methods have been used to deliver warm fluids to the surgical patient. These methods can be grouped into (a) those devices or methods which warm the fluid en route to the patient, and (b) those methods or devices which warm the fluid prior to administration. In some instances, these methods have been combined with varying degrees of effectiveness.

In all methods examined, there is an optimum delivery rate or range of rates at which the particular method is most effective. The slower infusion rates seem to be most susceptible to convective temperature loss from exposure to ambient room temperatures. This effect seems to be compounded when the infusion set is increased in length. Some studies find that prewarming fluids and delivering them through a short infusion set is the most effective means of delivering warmed fluids. Additionally, some find that insulating the fluid container

decreases the rate of heat loss and, therefore, increases the time the fluid can be administered at an effectively high temperature.

A number of questions remain. First, how effective is insulation in maintaining the temperature of prewarmed fluids before administration? Second, what is the minimum flow rate at which fluids can be administered, using the various methods, to maintain an adequate delivered temperature? Third, is there a safe, inexpensive, and simple method of delivering fluids to a surgical patient at or near body temperature?

Chapter Three

Methodology

Design

To answer the question, is there a difference between the Thermal Jacket™ and conventional means in maintaining the delivered temperature of prewarmed crystalloid solutions at various flow rates in the operating room environment, a quasi-experimental design was used. The independent variables--flow rate and temperature maintenance device--were manipulated while the dependent variable--delivered temperature--was observed. Time was treated as a fixed variable. There were three experimental groups and one control group. In the control group, no temperature maintenance device was employed.

Population and Sample

The population consisted of all Thermal Jacket™ insulation devices and all Hemokinetitherm® fluid warmers. The Thermal Jacket™ used for this study was obtained from the manufacturer and the Hemokinetitherm® was obtained from an operating room currently using this warmer. The Thermal Jacket™ was a quilted, wrap-around, insulation device which enclosed bags of intravenous fluid and was

secured by Velcro straps. The Thermal Jacket™ depicted in Figure 1 appears in a closed configuration.

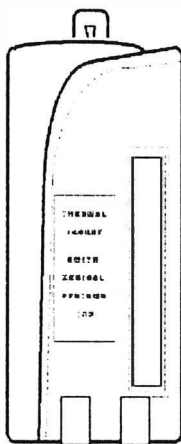


Figure 1. Thermal Jacket™ intravenous insulation device.

Treatment Groups

Group 1 was the control group. Intravenous (IV) fluids in this group were prewarmed and infused using no temperature maintenance device.

Group 2 consisted of prewarmed IV fluids infused through a water bath type blood warmer.

Group 3 consisted of IV fluids near 21 °C (ambient room temperature) and infused through a water bath type blood warmer.

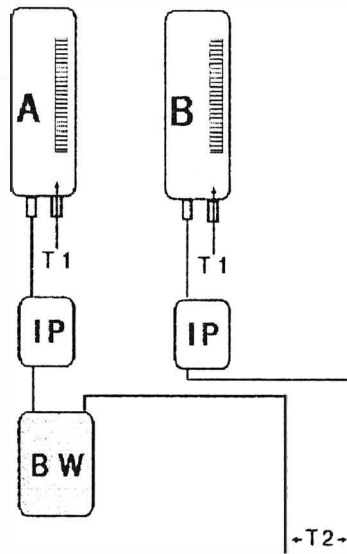
Group 4 consisted of prewarmed IV fluids infused using a Thermal Jacket™ insulation device.

Procedure

Attempts were made to replicate a typical operating room environment with the exception of a patient and the surgical team. Doors to the operating room were closed, room lights were on, and surgical lamps were lit and focused on the center of the operating room table which was located at approximately the center of the room. Ambient room temperature was set at the wall thermostat to 21 °C and measured throughout the study. All anesthesia equipment normally present in the room was turned on.

The blood warmers were placed near the head of and on opposite sides of the operating table and were filled with sterile water to the manufacturer's specifications. A 30 minute equilibration time was allowed prior to data collection to allow stabilization of the water bath temperature. The bags of fluid were hung at 200 cm from the floor and measures were taken to avoid contact between the bags, any part of the infusion sets, and other uninsulated surface or object. All infusion pumps were attached to IV poles 120 cm from the floor with a minimum distance of 50 cm between them. The infusion setup is depicted in Figure 2.

One liter Viaflex™ (Fenwall Laboratories, Division of Travenol Laboratories, Incorporated, 1 Baxter Parkway, Deerfield, Illinois, 60015.) plastic containers of lactated Ringer's solution used for Groups 1, 2, and 4 were prewarmed to approximately 42 °C by heating in a microwave oven set to maximum power for



A. Infusion set as used with Group 2 and Group 3.

B. Infusion set as used with Group 1 and Group 4.

IP. Infusion pump.

BW. Hemokinetitherm® Blood Warmer.

T1. Temperature measurement site inside the bag of intravenous fluid.

T2. Distal temperature measurement site.

Figure 2. Infusion setup.

two minutes. The same type fluid was stored at room temperature and used for group 3.

Identical standard intravenous pump tubing was used for all groups. Groups 2 and 3 incorporated a blood warming coil attached to the distal end of the pump tubing and immersed in a preheated blood warmer according to the manufacturer's specifications. The intravenous pump tubing and blood warming coils were not modified from their normal length. The distal end of each infusion set was secured with tape to a piece of foam rubber on the operating table 90 cm from the floor and allowed to drain into a collection container.

After a baseline measurement, temperatures were recorded at 10 minute intervals over a 60 minute period at 2 different sites for each group. The first site measured was in the lower portion of the IV solution bag. This was accomplished by inserting a Mon-A-Therm® temperature probe through the rubber medication port in the bottom of the container. The second site was 2 cm from the distal end of the infusion set and was measured using a Mon-A-Therm® inline thermistor. Temperatures were measured at flow rates of 100, 250, 500, 750, and 1,000 ml/hr for each group. Room temperature was recorded at the same time intervals, and to decrease any variation that room temperature may have had, all groups were run simultaneously at each flow rate.

Instrumentation

Fluid Warmer. The DUPACO Hemokinetitherm® Model #32400 Fluid Warmer was used. It is an automatic system regulated by a proportional

controller which, according to the manufacturer, maintains a precise temperature in the 550 ml water reservoir between 33 °C and 37 °C for the flow rates used in this study. (DUPACO, Incorporated, 1740 LaCosta Meadows Drive, San Marcos, California, 92069.)

Blood Warmer Coil. A Fenwal Blood Warmer Coil with Extension Set (#4C2403) was used. Fluid was infused through the warming coil which was immersed in the water reservoir of the DUPACO Hemokinetitherm® in accordance with the manufacturer's instructions. The Fenwal coil measures approximately 535 cm in total length and contains a volume of approximately 50 ml. Of the 535 cm, approximately 46 cm of the tubing is proximal to the coil and 174 cm (extension set) is distal to the coil. (Fenwall Laboratories, Division of Travenol Laboratories, Incorporated, 1 Baxter Parkway, Deerfield, Illinois, 60015.)

Infusion Pump. The Travenol Flo-Gard Model 8000 volumetric infusion pump was used. The Flo-Gard 8000 delivered from 1 ml/hr to 999 ml/hr in 1 ml/hr increments. It was accurate to ± 4 ml/hr at a setting of 125 ml/hr according to the manufacturer. (Travenol Laboratories, Incorporated, 1 Baxter Parkway, Deerfield, Illinois, 60015.)

Volumetric Pump Solution Set. The Travenol Volumetric Pump Solution Set (#2C1031) was used. The set measured approximately 270 cm in length and contained a volume of approximately 15 ml distal to the drip chamber. (Travenol Laboratories, Incorporated, 1 Baxter Parkway, Deerfield, Illinois, 60015.)

Temperature Monitoring System. The Mon-A-Therm® Model 6500 temperature monitoring system was used to measure temperatures at the two sites in the infusion setup and also to measure room temperature. The Mon-A-Therm® is a two-channel, microprocessor-based, instrument which provides temperature measurements with a resolution of 0.1 °C accurate to 0.1 °C over a range of 1-50 °C. It had a pre-set calibration which was checked each time the module was turned on. In this study, all temperature monitors used registered a calibration number of 100.0 ± 0.2 which indicated proper calibration according to the manufacturer's specifications. (Mon-A-Therm®, Incorporated, 520 South Jefferson Avenue, St. Louis, Missouri, 63103.)

Temperature Monitoring Probes. There were two types of temperature probes used in this study. A Mon-A-Therm®, 9 Fr., esophageal stethoscope with temperature sensor (Catalog #503-0032) was used to measure temperatures in the intravenous fluid containers. A Mon-A-Therm® luer lock temperature sensor (Catalog #503-0501) was used to measure delivered temperatures at the distal end of the infusion set. (Mon-A-Therm®, Incorporated, 520 South Jefferson Avenue, St. Louis, Missouri, 63103.)

Data Analysis

A two-way analysis of variance (ANOVA) was used to examine the effects of flow rate and the various methods of temperature maintenance on the delivered temperature of intravenous crystalloid solutions (group x flow rate). There were four levels of the group factor (Groups 1, 2, 3, 4) and five levels of the flow rate factor (100, 250, 500, 750, 1,000 ml/hr).

Chapter Four

Results

A quasi-experimental design was used to determine the effect of the Thermal Jacket™, compared to conventional means, on the delivered temperature of prewarmed, crystalloid, intravenous solutions in the operating room environment. One control group and three experimental groups were used. Group 1 was the control group and consisted of prewarmed intravenous fluids infused using no temperature maintenance device. Group 2 consisted of prewarmed IV fluids infused through a water bath type blood warmer. Group 3 consisted of IV fluids near ambient room temperature and infused through a water bath type blood warmer. Group 4 consisted of prewarmed IV fluids infused using a Thermal Jacket™ insulation device.

Ambient room temperature was measured at 21.0 ± 0.2 °C, but not controlled. Room temperature fluids were determined to be 22.05 ± 0.45 °C and prewarmed fluid were heated to 41.45 ± 1.05 °C. The mean temperatures of the fluid containers and the delivered temperatures for each group are shown in Table 1. The mean temperatures of both the fluid containers and the delivered temperatures for each flow rate are shown in Table 2. A Wilk's multifactor

Table 1

Temperature of Fluids from Container
to Distal Site by Group*

| Group | Temperature (° C) | |
|-------|--------------------|-------------|
| | Container | Distal Site |
| 1 | 36.7 ± 0.45 | 25.4 ± 0.54 |
| 2 | 37.3 ± 0.46 | 28.8 ± 0.61 |
| 3 | 21.9 ± 0.05 | 27.0 ± 0.52 |
| 4 | 39.9 ± 0.18 | 26.8 ± 0.65 |

* Mean Temperature ± S.E.M.

Table 2

Temperature of Fluids from Container
to Distal Site by Flow Rate^{*}

| Flow Rate (ml/hr) | Temperature (° C) | |
|----------------------|--------------------|-------------|
| | Container | Distal Site |
| 100 | 33.6 ± 1.40 | 22.7 ± 0.43 |
| 250 | 34.4 ± 1.40 | 25.1 ± 0.39 |
| 500 | 33.8 ± 1.41 | 27.4 ± 0.53 |
| 750 | 34.0 ± 1.43 | 29.4 ± 0.46 |
| 1,000 | 33.8 ± 1.40 | 30.4 ± 0.42 |

^{*} Mean Temperature ± S.E.M.

analysis of variance revealed a highly significant overall group effect, $F(6, 262) = 150.59$, $p < .0001$, as well as a highly significant flow rate effect, $F(8, 262) = 25.27$, $p < .0001$, on the delivered temperature.

Group Effect

Analysis of variance showed a highly significant group effect on the delivered temperature. A Bonferroni multiple comparisons test was used to determine which groups were the major contributors. Group 2 had a significantly higher delivered temperature ($p < .05$) than the other groups while Group 1 had a significantly lower delivered temperature ($p < .05$). There was no statistically significant difference between the delivered temperatures of Group 2 and Group 3.

Analysis of variance also showed a highly significant group effect on the temperature in the fluid container ($p < .05$). A Bonferroni multiple comparisons test was used to determine which groups were the major contributors to this effect. Group 4 had a significantly higher temperature than the other groups ($p < .05$) while Group 3 had a significantly lower temperature ($p < .05$). There was no significant difference between the fluid container temperatures in Group 1 and Group 2.

Flow Rate Effect

Analysis of variance showed a highly significant flow rate effect. A Bonferroni multiple comparisons test was used to determine the relationship

between flow rate and the delivered temperature. A statistically significant ($p < .05$) difference was found between flow rates of 100, 250, 500, and 750 ml/hr, with the higher flow rates resulting in higher delivered temperatures. There was no significant difference, however, between flow rates of 750 and 1,000 ml/hr. Analysis of variance showed no significant difference between the fluid container temperatures at the various flow rates.

Chapter Five

Discussion

The purpose of this quasi-experimental study was to determine the relationship between the flow rate and the delivered temperature of prewarmed, crystalloid, intravenous solutions when using the Thermal Jacket™ as compared to a conventional blood warming apparatus.

Hypothesis Testing

There were six hypotheses proposed in this study. They were as follows:

- 1) there will be no difference between the delivered temperatures of prewarmed and room temperature fluids using a blood warmer apparatus;
- 2) there will be no difference between the delivered temperatures of room temperature fluids using a blood warmer apparatus and prewarmed fluids using no temperature maintenance device;
- 3) there will be no difference between the delivered temperatures of room temperature fluids using a blood warmer apparatus and prewarmed fluids using the Thermal Jacket™;

4) there will be no difference between the delivered temperatures of prewarmed fluids using a blood warmer apparatus when compared with no temperature maintenance device;

5) there will be no difference between the delivered temperatures of prewarmed fluids using the Thermal Jacket™ when compared with no temperature maintenance device; and

6) there will be no difference between the delivered temperatures of prewarmed fluids using the Thermal Jacket™ when compared with a blood warmer apparatus.

Only one of these hypotheses failed to be rejected: there was no significant difference between the delivered temperatures of room temperature fluids passed through a blood warming device and prewarmed fluids using the Thermal Jacket™ (Hypothesis 3). The remaining five hypotheses were rejected. There was a significant difference between the delivered temperatures when comparing the other group combinations.

Correlation With Previous Studies

This study supports the findings of Baker (1985), Cooter (1987), and Rymers (1988) that faster flow rates resulted in less heat loss and a higher delivered temperature. Unlike Baker's findings, at no time did the delivered temperature fall below the initial temperature of unwarmed fluid. One explanation for this could be the care taken in avoiding contact between the infusion tubing and any uninsulated surface. There were no unexplained temperature fluctuations in this

study as in Cooter's and Rymers' studies. Flow rates were not as high in this study, therefore, only one infusion pump was used for each group.

This study also supported the findings of Norman et al. (1986). First, insulation of prewarmed fluids resulted in higher delivered temperatures than uninsulated fluids. Second, higher flow rates resulted in higher delivered temperatures due to a decrease in heat loss.

The delivered temperatures in this study did not match those of Boyan and Howland (1961). They reported delivered temperatures as high as 35 °C at flow rates of 3,000 ml/hr. At a flow rate of 1,000 ml/hr, room temperature fluid passing through the blood warmer reached a maximum temperature of 30.6 °C. This discrepancy could be due to the flow rates or types of fluid compared. Boyan and Howland used banked blood, whereas, this study used crystalloid fluid. At slower flow rates, the warmed fluid is exposed to colder ambient room air longer and would tend to lose more heat.

The results of this study differed from those of Aldrete (1985). Aldrete found that preheating, alone, resulted in the highest delivered temperature while the combination of preheating and blood warmer resulted in a still higher delivered temperature than the blood warmer alone. This study found that the combination of preheating and infusion through a blood warmer resulted in the highest delivered temperature. Infusing room temperature fluid through a blood warmer resulted in temperatures similar to the combination of preheating and using the Thermal Jacket™. The obvious difference between the studies was the length of infusion tubing. In Aldrete's study the length of tubing distal to the

blood warmer was nearly the same length as the straight infusion set used. In the current study, the straight tubing was 96 cm longer than the tubing distal to the blood warmer. As Rymers (1988) found, the length of tubing contributes significantly to the amount of heat loss. If the tubing lengths were identical, the results may have more closely correlated with Aldrete's.

Aldrete also reported that the fluid in the container cooled more per volume infused at slower infusion rates. The problem of this reporting was pointed out previously, it did not account for the length of time the fluid was exposed to room air. In the current study, at slower infusion rates, there was a larger amount of heat lost per volume infused (0.086°C per ml at 100 ml/hr compared to 0.007°C at 1000 ml/hr) supporting the earlier criticism, there was no significant statistical correlation between infusion rates and temperature of fluid in the container in this study.

Compared to Fried's (1986) study, the methods used in this investigation were far inferior in maintaining a warm delivery temperature. The difference was possibly related to the equipment used. Fried used an extracorporeal heat exchanger with a large countercurrent waterflow as the heat source. This was a much more elaborate and expensive setup, and was designed as a rapid solution administration set for use with mass trauma cases. The methods used in the current study correspond to more widely used operating room techniques.

Flow Rate Effect

There was a highly significant correlation between flow rate and the delivered temperature. Higher flow rates resulted in higher delivered temperatures due, at least in part, to the time the fluid was exposed to the relatively cooler, ambient room air. There was an insignificant correlation between flow rate and the temperature of the fluid in the container.

Group Effect

There was a highly significant difference between the various groups in maintaining the temperature of the fluid in the container as well as the delivered temperature. The Thermal Jacket™ provided insulation and maintained a higher temperature of the fluid in the container. The combination of prewarming and delivery through a blood warmer resulted in higher delivered temperatures at all flow rates. Prewarming fluids and insulating with the Thermal Jacket™ provided delivery temperatures similar to those of infusing room temperature fluids through a blood warmer and through a conspicuously shorter length of tubing. Prewarming and infusing through a straight infusion set resulted in the lowest delivered temperatures.

Limitations

The only serious limitation to this study was the length of tubing used with the various groups. The tubing lengths were chosen to resemble, as closely as

possible, the clinical situation in which these methods of warming fluids might be utilized. Various blood warmers have differences in their effectiveness in warming fluids over a range of flow rates. This, combined with the broad range of temperatures at which the fluid can be prewarmed, varying ambient room temperatures, and combinations of other heat-maintenance maneuvers, limits the generalizability of this study.

Recommendations for Further Study

The study should be repeated with the following modifications:

- 1) Alter the length of the infusion tubing of the straight infusion set to match that distal to the blood warmer.
- 2) Vary room temperature to both extremes to quantify the room temperature effect on heat loss.
- 3) Manipulate the flow rates over a larger range.
- 4) Insulate the infusion tubing to further decrease heat loss to ambient room air.

The Thermal Jacket™ should also be investigated in the clinical setting. Patients could be divided into 3 groups. One group could have room temperature fluids infused throughout their surgery. Another group could have fluids delivered via a conventional blood warming device. A third group could have fluids prewarmed and delivered using the Thermal Jacket™.

Conclusion

In the setting studied, the Thermal Jacket™ was an effective means of delivering prewarmed, crystalloid, intravenous fluid via a straight infusion set. This could be an advantage in situations where a blood warming device may not be available or the high resistance to flow in the blood warming coil may impede administration of fluids at desired flow rates. Though using the blood warmer with prewarmed fluids was more effective in this study, this difference may be reduced by shortening the length of intravenous tubing used in conjunction with the Thermal Jacket™. Additional studies of the Thermal Jacket™, as outlined, may clarify the advantages of each method.

References

References

- Aldrete, J. A. (1985). Preventing hypothermia in trauma patients by microwave warming of IV fluids. The Journal of Emergency Medicine, 3, 435-442.
- Baker, D. R. (1985). The correlation of flow rate and heat loss of warmed intravenous fluids. Unpublished master's thesis, Virginia Commonwealth University, Richmond, Virginia.
- Berne, R. M., & Levy, M. N. (Eds.). (1983). Physiology. St. Louis: Mosby.
- Boyan, C. P., & Howland, W. S. (1961). Blood temperature: A critical factor in massive transfusion. Anesthesiology, 22, 559-563.
- Boyan, C. P., & Howland, W. S. (1962). Problems related to massive blood replacement. Anesthesia and Analgesia, 41, 497-507.
- Boyan, C. P., & Howland, W. S. (1963). Cardiac arrest and temperature of banked blood. Journal of the American Medical Association, 183, 144-146.
- Buckhold, D. K., Pryor, W. H., Homer, L. D., Uddin, D. E., Hickey, T. M., & DiSimone A. G. (1978). Hemorrhagic shock treatment with hot intravenous fluid in dogs. The Journal of Trauma, 18, 726-729.
- Cabanac, M. (1975). Temperature regulation. Annual Review of Physiology, 37, 415-439.
- Churchill-Davidson, H. C. (1955). Hypothermia. British Journal of Anaesthesia, 27, 313-319.
- Conahan, T. J., Williams, G. D., Apfelbaum, J. L., & Lecky, J. H. (1985). Airway heating reduces recovery time (cost) in outpatients. Anesthesiology, 63, A166.
- Cooter, R. L. (1987). The effect of flow rate on the temperature of packed red blood cells passing through a fluid warmer. Unpublished master's thesis, Virginia Commonwealth University, Richmond, Virginia.

- Copping, J. W., Mather, G. S., & Winkler, J. M. (1972). Physiological responses to the administration of cold, room temperature, and warm balanced salt solutions in hemorrhagic shock in dogs. Surgery, 71, 206-209.
- Downey, J. A., Miller, J. M., & Darling, R. C. (1969). Thermoregulatory responses to deep and superficial cooling in spinal man. Journal of Applied Physiology, 27, 209-212.
- Elder, P. T. (1984). Hypothermia: Anesthetic considerations. Anesthetic Update Series, 6, lesson 7.
- Flacke, W. E., Flacke, J. W., Ryan, J. F., & Britt, B. A. (1983). Altered temperature regulation. In F. Orkin & L. Cooperman (Eds.), Complications in Anesthesiology (pp. 277-313). Philadelphia: J. B. Lippincott.
- Fried, S. J., Satiani, B., & Zeeb, P. (1986). Normothermic rapid volume replacement for hypovolemic shock: An in vivo and in vitro study utilizing a new technique. The Journal of Trauma, 26, 183-188.
- Goldberg, M. E., Jan, R., Gregg, C. E., Berko, R., Marr, A. T., & Larijani, G. E. (1988). The heat and moisture exchanger does not preserve body temperature or reduce recovery time in outpatients undergoing surgery and anesthesia. Anesthesiology, 68, 122-123.
- Goldberg, M. J., & Roe, C. F. (1966). Temperature changes during anesthesia and operations. Archives of Surgery, 93, 365-369.
- Goudsouzian, N. G., Morris, R. H., & Ryan, J. F. (1973). The effects of a warming blanket on the maintenance of body temperatures in anesthetized infants and children. Anesthesiology, 39, 351-353.
- Hall, G. M. (1978). Body temperature and anaesthesia. British Journal of Anaesthesia, 50, 39-44.
- Harnett, R. M., Pruitt, J. R., & Sias, F. R. (1983). A review of the literature concerning resuscitation from hypothermia: Part II- selected rewarming protocols. Aviation, Space, and Environmental Medicine, 54, 487-495.
- Haslam, K. R., & Nielsen, C. H. (1986). Do passive heat and moisture exchangers keep the patient warm? Anesthesiology, 64, 379-381.
- Holdcraft A., & Hall, G. M. (1978). Heat loss during anaesthesia. British Journal of Anaesthesia, 50, 157-164.
- Howland, W. S., Boyan, C. P., & Schweizer, O. (1956). Ventricular fibrillation during massive blood replacement. American Journal of Surgery, 92, 356-360.

- Howland, W. S., Schweizer, O., Boyan, C. P., & Dotto, A. C. (1955). Physiologic alterations with massive blood replacement. Surgery, Gynecology, & Obstetrics, 101, 478-482.
- Lilly, R. G. (1986, October). Inadvertent hypothermia: A real problem. Paper presented at the American Society of Anesthesiologists Annual Meeting, 37th Annual Refresher Course Lectures and Clinical Update Program, Las Vegas.
- Little, D. M. (1959). Hypothermia. Anesthesiology, 20, 842-877.
- Lonning, P. E., Skulberg, A., & Abyholm, F. (1986). Accidental hypothermia. ACTA Anaesthesiologica Scandinavica, 30, 601-613.
- Morris, R. H. (1971). Operating room temperature and the anesthetized, paralyzed patient. Archives of Surgery, 102, 95-97.
- Morris, R. H., & Kumar, A. (1972). The effect of warming blankets on maintenance of body temperature of the anesthetized, paralyzed adult patient. Anesthesiology, 36, 408-411.
- Morris, R. H., & Wilkey, B. R. (1970). The effects of ambient temperature on patient temperature during surgery not involving body cavities. Anesthesiology, 32, 102-107.
- Newman, B. J. (1971). Control of accidental hypothermia. Anaesthesia, 26, 177-187.
- Norman, E. A., Ahmad, I., & Zeig, N. J. (1986). Delivery temperature of heated and cooled intravenous solutions. Anesthesia and Analgesia, 65, 693-699.
- Papenburg, R. R., English, M., Foot, E., Farias, R. T., & Hinchey, E. J. (1987). The effect of warm I.V. solutions on the management of surgical hypothermia. Anesthesiology, 67, A479.
- Pickering, B. G., Bristow, G. K., & Craig, D. B. (1977). Core rewarming by peritoneal irrigation in accidental hypothermia with cardiac arrest. Anesthesia and Analgesia, 56, 574-577.
- Roe, C. F., Goldberg, M. J., Blair, C. S., & Kinney, J. M. (1966). The influence of body temperature on early postoperative oxygen consumption. Surgery, 60, 85-92.
- Roizen, M. F., L'Hommedieu, Y. J., Wylie, C. S., & Ota, M. K. (1980). Operating room temperature prior to surgical draping: Effect on patient temperature in recovery room. Anesthesia and Analgesia, 59, 852-855.

- Rymers, S. J. (1988). The effect of flow rate and intravenous tubing length on the temperature of warmed packed red blood cells. Unpublished master's thesis, Virginia Commonwealth University, Richmond, Virginia.
- Scott S. M. (1967). Thermal blanket injury in the operating room. Archives of Surgery, 94, 181.
- Shanks, C. A. (1975). Control of heat balance during arterial surgery. Anaesthesia and Intensive Care, 3, 118-121.
- Stone, D. R., Downs, J. B., Paul, W. L., & Perkins, H. M. (1981). Adult body temperature and heated humidification of anesthetic gases during general anesthesia. Anesthesia and Analgesia, 60, 736-741.
- Vale, R. J. (1973). Normothermia: Its place in operative and postoperative care. Anaesthesia, 28, 236-252.
- Zorko, M. F., & Polsky, S. S. (1986). Rapid warming and infusion of packed red blood cells. Annals of Emergency Medicine, 15, 907-910.

Vita

