USE OF A STOCKINET CAP ON PREMATURE INFANTS AFTER DELIVERY

A THESIS

SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS

FOR THE DEGREE OF MASTER OF SCIENCE

IN THE GRADUATE SCHOOL OF THE

TEXAS WOMAN'S UNIVERSITY

COLLEGE OF NURSING

BY

JENIFER ROBERTS, B.S.N.

DENTON, TEXAS

AUGUST 1981

The Graduate School

Texas Woman's University

Denton, Texas

April 13 1981

We hereby recommend that the thesis prepared under our supervision by Jenifer Roberts entitled Use of a Stockinet Cap on Premature Infants After Delivery

be accepted as fulfilling this part of the requirements for the Degree of Master of Science.

Dissertation/Theses signature page is here.

To protect individuals we have covered their signatures.

ACKNOWLEDGMENTS

To my committee chairman, Judith Erlen, Ph.D., I wish to express my sincere appreciation for her helpful guidance and assistance during the preparation of this thesis. A special thanks for her careful supervision and attention to details.

To my committee members, Lois Hough, Ph.D., and Gail Watson, M.S., my appreciation for their helpful and scholarly comments during the formation of this thesis.

To Britt Canada, I wish to acknowledge his patience and expertise in the statistical analysis of the data.

To the labor and delivery and nursery staff at Parkland, a warm appreciation for their cooperation.

To my typist, Becky Shelton, many thanks for a job well done.

A special thanks are due to my parents for their continued support and encouragement. Without their help, emotionally and financially, this thesis would not have been written.

TABLE OF CONTENTS

Chapter

LIST OF TABLES

CHAPTER 1

INTRODUCTION

In the early 1900s, the importance of maintaining an infant's body temperature began to be more intensely studied by researchers concerned with excellence in the care of the newborn (Oliver, 1977). The fact was documented that newborn infants lose body heat to the surrounding environment if agressive precautions are not taken. The premature infant with his unique body structure and other physical characteristics is especially prone to lose body heat to the cooler environment. This process of heat loss is a stressor to the newly born infant and causes a metabolic reaction to take place which should be prevented, especially in the premature infant.

Nurses have used many different techniques and types of equipment to help maintain an infant's temperature within a thermoneutral range. One method that is employed forms a prototype for this study. This method consists of using a stockinet cap on the heads of premature infants who are being weaned from their incubators to open bassinets. The cap has been found adequate under most circumstances to maintain a premature infant's temperature within a thermoneutral range. Given that this method works on premature infants at one phase in their development, would this same technique work at an earlier time when premature infants experience their greatest exposure to cold stress, namely right after delivery?

Problem of Study

The problem of this study was to determine if the use of a stockinet cap would affect heat loss in premature infants from birth until admission to the nursery.

Justification of Problem

Korones (1972) stated that provision of an optimal thermal environment is one of the most important aspects of effective nursing care of the newborn due to the effect cold has on a newborn infant. There are numerous reasons why cold stress should be kept at a minimum, especially for the premature infant. Around the turn of the 20th century, Budin established that premature infants have a better survival rate if they are kept warm (Besch, Perlstein, Edward, Keenan, & Sutherland, 1971; Oliver, 1977). Warmth is essential for conserving energy and promoting the infants' growth and weight gain (Eoff, Meier, & Miller, 1974; Glass, Silverman, & Sinclair, 1968, Korones, 1972). In addition, provision of a thermoneutral environment for the newborn results in a more satisfactory recovery from birth asphyxia

(Adamsons & Towell, 1965; Gandy, Adamsons, Cunningham, Silverman, & James, 1964; Schultz, Soltész, Molnár, & Mestyán, 1979). Cooling of an infant also results in an increased metabolic rate, thereby increasing oxygen consumption (Brück, 1961, Taylor, 1971). Ziegal and Van Blarcom (1972) pointed out that chilling a baby after birth interferes with his ability to produce surfactant which is of particular importance in proper lung function in premature babies.

Physicians and nurses are aware of the numerous ill-effects of cold on the newborn, especially the premature infant. An infant loses heat by four modalities: conduction, convection, radiation, and evaporation. Conduction refers to transference of heat by direct contact with a cooler object, such as placing an infant on a cold metal scale. Convection refers to heat lost to cooler air blowing across the infant. Radiation is the transfer of heat through air or a vacuum with direct contact not being necessary. An example is heat being lost to a cool incubator wall that is next to a cool window. Evaporation is the cooling that occurs when body heat is used to vaporize a liquid such as amniotic fluid (Porth & Kaylor, 1978). In the delivery room, the infant is subject to heat loss from all four of these modalitites, but "heat loss due to radiation and convection together is twice that from evaporation" (Dahm & James, 1972, p. 512).

The premature infant is more at risk for heat loss than the full-term infant due to the unique characteristics of the premature infant. Adamsons (1966) has stated that thermal stability is mainly a function of body size, amount of surface area, amount of thermal insulation and the degree to which heat output can be increased. The premature is at risk in all of these categories. The infant has a small body size and a relatively large surface area with which to discharge heat to the surrounding environment. He has a deficient amount of subcutaneous fat to serve as insulation. While heat production is functional from the onset of life, it is insufficient to counterbalance the heat lost (Brück, 1961). Brück (1961) in his research in the area of thermoregulation in the newborn determined that

> heat loss per surface unit is 40% greater for newborn infants than for adults. . . . We may expect a heat loss in full-term infants about four times as great per body weight unit as in adults, and in premature infants (1,500 gm) at least 4.9 times as great as in adults. (pp. 103-104)

This phenomenon is primarily due to the fact that at birth the full-term infant's body weight is only 5% of adult body weight, but his surface area is 15% of the adult's. "This discrepancy between weight and body surface area is even greater for low birth-weight infants" (Korones, 1972,

p. 60). Because the body surface area is relatively large in comparison with weight, the infant, especially the premature infant, has greater opportunity to lose heat to the environment. Another reason the premature infant is more at risk to lose heat is the characteristic posture of the premature that results in increased surface area. A full-term infant characteristically keeps the extremities flexed, thereby decreasing the amount of surface area that is available for heat exchange. The premature infant due to neurologic immaturity keeps the extremities extended providing a larger surface area for heat exchange (Oliver, 1977).

In recent years, radiant warmers were developed to provide a source of external heat that would allow procedures and observations to take place in an open environment. Use of a radiant warmer, in addition to drying the infant, has proved to be effective in maintaining the full-term infant's body heat. The full-term infant has little problem maintaining body temperature, while a premature infant often has lost body heat before arrival in the nursery (Dahm & James, 1972; Du & Oliver, 1969; Rosen, Dupuis, & Wichman, 1978).

In the researcher's experience, a large number of newly born premature infants have been identified as being hypothermic on admission to the nursery. A study resulting in similar observations was done by three nurses who were concerned with the thermoneutral conditions in the delivery room at their hospital. They compared the first three rectal temperatures taken on infants born vaginally and by Cesarean section. The researchers concluded:

> that the present methods of thermoregulation in the delivery room seem satisfactory for infants with adequate brown fat deposits, but that infants who are small for dates or premature need special care to ensure heat preservation. Aware of their problems, delivery room staff can prepare better for the delivery of these infants. (Rosen et al., 1978, p. 1693)

This present study was undertaken to determine if the use of a stockinet cap immediately after delivery as an additional nursing measure would help maintain a premature infant's body temperature within a thermoneutral range.

Conceptual Framework

Neuman's (1974) health-care systems model, "total person approach to patient problems" (p. 99), has designed a model for nurses to use as a guide for care and services aimed at assisting an individual or group in attaining and maintaining a maximum level of wellness. The nurse's function is directed toward reducing the stress factors and any adverse conditions that may interfere with optimal functioning in a given client situation. Neuman (1974) continued by emphasizing that the current trend in health care is toward primary prevention. She defined primary prevention as relating to "general knowledge that is applied to individual patient assessment in an attempt to identify and allay the possible risk factors associated with stressors" (p. 101).

In the model, Neuman (1974) conceptualized man as a series of concentric circles with the innermost circle representing the basic survival factors such as normal temperature range, one's genetic structure, individual response pattern, the strength and weakness of various body parts, and ego structure. The surrounding larger circles represent the individual's lines of resistance, which are the person's "internal set of resistance . . . factors which attempt to stabilize and return him to his normal line of defense should a stressor break through it" (p. 101). The outermost circle, a broken line, is the flexible line of defense which signifies one's ability to ward off stressors.

This flexible line of defense serves as a buffer for the normal line of defense which is a person's steady state developed over time. The flexible line of defense can be weakened by periods of under-nutrition and lack of sleep and strengthened by proper exercise and good nutrition.

Any stressor has the potential of incapacitating or reducing the integrity of the lines of resistance, thereby lessening one's ability to cope with any further stressors which might come along (Neuman, 1974). Selye (1956) previously had noted this phenomenon in his research on stress. In defining the stages of the stress syndrome, Selye discussed the stage of resistance saying that:

> The stage of resistance received its name because here, resistance to the particular agent, which produced this stage of the (general or local) adaptation syndrome, is at its peak; yet at the same time, resistance to most other agents tends to fall below normal. (p. 227)

Thinking in terms of Neuman's (1974) model, this would mean that if an individual is exposed to numerous stressors, or agents in Selye's terms, at any one time, there is increased possibility that the lines of resistance and the lines of defense would be broken, thereby causing a threat to the innermost basic structure.

In Neuman's (1974) model, intervention can occur any time a stressor is suspected or identified. Prevention can be either primary, secondary, or tertiary. "Primary prevention relates to general knowledge that is applied to individual patient assessment in an attempt to identify and allay the possible risk factors associated with stressors" (Neuman, 1974, p. 101). Prevention is secondary when intervention begins after symptoms have been identified. Tertiary prevention is the rehabilitation process beginning after active treatment has begun and some degree of stability has been reached.

"The health-care systems model is intended to represent an individual who is subjected to the impact of stressors" (Neuman, 1974, p. 101). The newborn infant, especially the premature infant, is an individual who is subject to the impact of many stressors such as cold, physical sensations, nursing procedures, and changes in body position. This infant defends his inner basic structure by his flexible and normal lines of defense and his lines of resistance.

The infant's normal line of defense is the layer of subcutaneous fat (generally known to be inadequate in premature infants), posture, normal level of vasoconstriction, and normal rate of metabolism. The infant's flexible line of defense would refer to the general condition at birth, and would vary depending on such factors as oxygenation, vigor, nourishment, and metabolic state. When the stressor cold penetrates the normal line of defense in a premature infant, the infant reacts by activating the lines of resistance, which means increasing the metabolic rate, increasing vasoconstriction, and mobilizing brown fat.

The nursing care given to newborns as well as all other patients should be aimed at primary prevention. Neuman (1974), in talking of primary prevention stated:

> The "actor" or intervenor would perhaps attempt to reduce the possibility of the individual's encounter with the stressor or in some way attempt to strengthen the individual's flexible line of defense to decrease the possibility of a reaction. (p. 104)

Consequently, the addition of a cap to a premature infant after delivery should aid in prevention of cold stress by (a) reducing the possibility of the infant's encounter with the stressor cold by reducing the surface area available for heat exchange and (b) strengthening the infant's flexible line of defense by providing increased insulation from the cold, thereby preventing or decreasing initial loss of body heat.

Assumptions

The following assumptions were made:

1. It is desirable to maintain a newly born premature infant's body temperature within a thermoneutral range.

2. Since an infant's head is a large portion of his total body surface area, he loses a significant amount of heat through his scalp.

3. Primary prevention of penetration of the stressor cold is preferable to secondary prevention.

Hypotheses

The hypotheses for this study were:

1. There will be no difference on arrival in the nursery in the admission axillary temperatures of premature infants who receive a stockinet cap after delivery and premature infants who do not receive a cap after delivery.

2. There will be no difference in the amount of heat lost from the time the axillary temperature is obtained in the delivery room until the axillary temperature is obtained in the nursery in premature infants who receive a stockinet cap after delivery and premature infants who do not receive a cap after delivery.

Definition of Terms

For the purposes of this study the terms used herein were defined as follows:

1. Arrival in the nursery--a lapse of no more than 10 minutes after the infant arrived in the nursery from the delivery room.

2. Cap--a sterile headpiece made of stockinet (a material used to protect the skin under orthopedic casts) which covers the forehead, the ears, underneath the occipital bone, and all the area above this plane.

3. Axillary temperature--the temperature obtained by using an IVAC electric thermometer in the test mode held under the right arm for 5 minutes.

4. Amount of heat lost--the temperature difference between the axillary temperature obtained in the delivery room and the axillary temperature obtained in the nursery.

Limitations

The variations present in the study over which the researcher had no control were:

1. The time at which the umbilical cord was clamped.

2. The amount of time between birth and arrival in the nursery.

3. The use of drugs which may cross the placental barrier that were given to the mother prior to delivery.

4. The temperature and humidity in the delivery room, halls, elevator, incubator, and nursery.

5. The amount of time that any body part was exposed to air prior to complete delivery.

6. The time the initial axillary temperature was taken in the delivery room.

7. The temperature of the radiant warmers.

8. The infants who had blood drawn for culture.

9. Whether or not the premature infants were hand carried or transported in incubators to the nursery.

10. The infants who received oxygen by face mask after delivery.

Summary

This chapter presented an introduction to the problem of heat loss in premature infants. The problem of this study was: to determine if the use of a stockinet cap would have an effect on heat loss in premature infants from birth until admission to the nursery. Findings from literature and the researcher's experience were used to support the importance of this problem to nurses caring for premature infants. Neuman's model was outlined as a conceptual framework for the study. The assumptions made were stated along with the hypotheses. The terms used in this study were defined and the limitations stated.

CHAPTER 2

REVIEW OF LITERATURE

This chapter includes studies related to thermoregulation. The chapter begins with a brief synopsis of progress made in thermoregulatory knowledge and techniques used to prevent hypothermia in newborns since the early 1900s. Studies are cited that show both full-term and premature infants to be at risk to lose body heat. The next topic deals with the consequences heat loss has for a premature infant. Various methods and techniques of maintaining body temperature in newborns are discussed followed by some studies that correlate skin temperatures with increased metabolic rate. Studies are then presented on the various types of thermometers available.

Historical Perspective

There has been much interest in the whole area of thermoregulation in the newborn, particularly in the last 80 years. Questions regarding what is the ideal temperature for a newborn infant, what effects do different temperatures have on newborn infants, what are normal ranges of temperatures, how to keep infants in a thermal neutral environment, and many others of similar nature have been the subject of research. Budin, historically the first neonatologist, brought the importance of temperature regulation in newborns to the notice of other researchers with his book, Le Nourisson (The Nursling), published in the early 1900s. Budin (cited by Oliver, 1977) found that infants weighing less than 2,000 gm had a mortality rate of 90% if the body temperature was allowed to fall between a range of 32-33.5°C. This mortality rate increased to 98% if the temperature fell below 32°C. If the temperature was kept above 33.5°C the mortality rate was substantially reduced to 23% in infants of comparable size. "Sadly his [Budin's] observations were neither fully understood nor appreciated in the first fifty years of this century" (Klaus & Fanaroff, 1973, p. 58).

Oliver (1977) continued to outline the progress made in improving thermal conditions for newborn infants by showing changes in care brought about by results of research done in this area. In the early part of this century, low birth-weight infants were heavily swaddled and placed in open heated cribs as designed by Hess, a neonatologist. Cooney, in the 1930 Chicago Exposition, exhibited low birth-weight infants in these open incubators.

Later, enclosed, convection-heated incubators were designed with the babies lying unclothed in them at arbitrarily selected temperatures ranging between 28.9°C and 31.6°C. In the early 1950s, some research was done without control groups that reported demonstrating an advantage in keeping premature infants in lower ambient temperatures that resulted in hypothermia. In 1958, Silverman, Fertig, and Berger clearly demonstrated that infants kept in a higher ambient temperature of 31.6°C had an improved survival rate. Two studies (Day, Caliguiri, Kamenski, & Ehrlich, 1964; Buetow & Klein, 1964) were done which showed that if infants weighing less than 1,750 gm were placed in a convection-heated incubator at 31.6°C plus an infrared heat input that would keep the skin at 36°C, the mortality of these infants would be significantly less than infants placed in incubators at 31.6°C without the infrared heat input.

> It is clear that optimal thermal conditions are important to survival and that an understanding of temperature regulation and its clinical correlations is essential in the management of the newborn. (Behrman, 1977, p. 224)

Infants are at Risk to Lose Heat

Providing a thermal neutral environment is one of the major concerns in providing care to a newborn. This is primarily the responsibility of the neonatal nurse. Korones (1972) has stated that,

> Maintenance of an optimal thermal environment is one of the most important aspects of effective neonatal care, and without knowledge of the basic principles of thermoregulation a nurse cannot provide it consistently. However, with the facts at her command she can prevent cold stress and can solve some of the many riddles that arise daily. (p. 58)

Williams and Lancaster (1976) expressed concern over the growing number of babies being exposed to cold stress in spite of the fact that hypothermia had been associated with metabolic abnormalities and increased neonatal mortality. They hypothesized that the problem could be due to a lack of understanding of the basic principles of thermoregulation along with a failure to apply these principles maximally in neonatal care.

Brück (1961) in his research on thermoregulation in the newborn, established that newborn infants were at risk to lose body heat and estimated that this risk was proportionally greater in infants than in adults. He determined that heat loss in the newborn infant was four times that of an adult per unit of body weight. Adamsons (1966) attributed the cause of this heat loss in infants to their physical makeup. He stated that infants have a high thermal conductance level which is due to their lack of subcutaneous fat. This deficiency facilitates the loss of the infant's body heat to the surrounding environment.

Sinclair (1975) also underlined the importance of subcutaneous fat in maintaining body temperature by stating:

> Moreover, the low thermal insulation of the newborn infant (chiefly the result of small body size) confers a limited thermoregulatory range, so that when exposed to cold, the baby may show a profound fall in body temperature despite his vigorous metabolic response. (p. 147)

Another major reason that the infant loses heat is due to the fact that he has a small body size with a comparatively large body surface area. Korones (1972) said that heat loss is basically a function of the ratio between body surface and body weight. "The larger the surface in relation to body mass, the greater the opportunity to dissipate heat to the environment" (p. 60). Adamsons (1966) stated that at birth, the body mass of a newborn infant is about 5% that of the adult, whereas his body surface area amounts to nearly 15%. This larger body surface area provides greater opportunity for the infant to lose body heat to the environment. Oliver (1965) also noted that infants tended to become hypothermic because of their greater body surface area and increased tissue conductivity which he attributed to their lack of subcutaneous fat. Brück (1961) estimated this heat loss per surface unit to be 40% greater in newborn infants than in adults.

Body temperature is not only related to the amount of heat lost but also on the ability of the individual to produce heat (Heim, 1971). The question becomes, does an infant have the ability to produce heat? At one time infants were thought to be poikilothermic, which means that their body temperature becomes the same as the environmental temperature, that they make no effort to regulate their own temperature (Adamsons, 1966). More recent findings have determined that newborns are homiothermic, which means that they increase oxygen consumption to keep their deep body temperature relatively constant within limits when they are exposed to temperatures below their thermal neutral range (Heim, 1971). In comparison to the adult, newborn infants have a low rate of heat production in relation to their surface area, but a high rate for their body weight (Sinclair, 1971). Korones (1972) pointed out that thermal stability is more precarious in newborns than in older individuals due to the large amount of heat they lose and not to their ability to produce heat. Brück (1961) found that, in comparison with adults, the metabolic rise that occurs in infants in response to cooling is large, even during the first day of life. If a newborn is put in an environmental temperature of 23°C, there is a 100% rise in the metabolic rate. An adult in 23°C may show no rise in metabolic rate or at the most an increase of 25%

(Thauer & Wezler, 1943; Göpfert & Stufler, 1952). Adams, Fujiwara, Spears, and Hodgman (1964) found the infant capable of producing heat at the time of birth. Although measurements were not made within the first few minutes after birth, heat production was demonstrated at the age of 15 minutes.

Although infants are capable of producing heat, this heat production is a compensatory reaction and cannot function for an indefinite period of time.

> The neonate subjected to severe or protracted periods of cold stress will be unable to maintain a normal body temperature because the heat the baby loses will exceed the heat the baby can produce. (Williams & Lancaster, 1976, p. 356)

A newborn can produce heat, but there is an amount and a duration beyond which the infant's capacity for heat production is overcome by the amount of heat lost.

One of the major threats imposed on an infant's thermoregulatory ability is the thermal conditions present in the average delivery room. Sinclair (1975) noted that the average delivery room temperature is 23^{o}C which is midway in the thermal comfort zone for lightly-dressed adults. This temperature, however, is more than 10^{o}C below the thermal neutral environment for the infant. Due to the large radiant, convective and evaporative losses from the warm, wet skin of the newborn, the infant's temperature initially falls 2-3^{o}C. This fall in temperature corresponds to an energy expenditure of approximately .2 calories per kilogram per minute in the first few minutes after birth (Adamsons & Towell, 1965). Adamsons (1966) stated that this drop corresponds with a rate of fall of deep body and skin temperature of about .1°C per minute and .3°C respectively, or a heat loss of approximately 200 calories per kilogram per minute. Maximal heat production in an adult male is 90 calories per kilogram per minute. In comparing the amount of heat lost by a newborn infant in the delivery room with the maximal amount of heat produced in an adult male, "it is evident that body temperature of the newborn baby would fall even if heat production per unit of body mass were twice that of an adult" (Adamsons, 1966, p. 605). Ziegal and Van Blarcom (1972) stated that the average infant's body temperature is lowered somewhere between 33.3°C and 34.4°C shortly after birth. Sinclair (1975) found the average body temperature to be between 34.8°C and 35.8°C. Dahm and James (1972) stated that body temperature begins to fall after birth even if efforts are made to conserve heat by placing the infant under a radiant warmer. These authors found that when an infant is dried and placed under a radiant warmer, he loses about 22.5 calories per kilogram per minute over the first 30 minutes of life. North (1961) found that the average amount of heat lost during the interval between birth and arrival in the nursery to be between two and four degrees farhenheit. These studies have demonstrated that the infant is at great risk to lose heat in the average delivery room.

There are several other factors that either interfere with an infant's heat production, or affect the amount of heat lost, thereby having an effect on the infant's thermal stability. Washington (1978) stated several conditions that can result in hypothermia or some variation in the infant's ability to regulate his body temperature. These conditions were sepsis, central nervous system lesions, birth asphyxia, and intracranial hemorrhage. Adamsons (1966) also listed hypoxia, drugs, hypotension, hypercapnia, and hypoglycemia as interfering with thermal homeostasis.

There are several groups of drugs that have been found to affect temperature regulation in the adult. These include the hypnotics, analgesics, antipyretics, anes-thetics, and hormones. Burnard and Cross (1958) showed that while meperidine did not affect the temperature of the mother, the infant whose mother received meperidine showed a distinct drop in body temperature for the next 20 hours compared to an infant whose mother did not receive any meperidine during labor. Oliver (1977) stated that Diazepam given to the mother during labor also affects the thermoregulatory response in a newborn exposed to cold.

Lack of oxygen has been cited as affecting temperature regulation. Heim (1971) stated that under hypoxic conditions heat production is suppressed by some mechanism that has not been clearly identified. This suppression has been demonstrated in newborn rabbits. As soon as the newborn rabbits breathe 10% oxygen, the temperature over their brown fat deposits drops sharply indicating a drop in heat production (Heim & Hull, 1966). Similarly, Heim and Kellermayer, as cited in Heim (1971), performed a postmortem study of four human babies with congenital heart disease. They found that the brown fat tissue was full of fat droplets in spite of the fact that the babies were malnourished because of their disease and were cared for in ambient temperatures below their thermoneutral zone. Heim (1971) hypothesized that these infants were unable to utilize their brown fat due to the hypoxic conditions imposed by their disease. A synthesis of these studies suggests hypoxia may suppress the ability of an infant to produce heat.

Another process that causes an alteration in the normal process of temperature regulation in the newborn is blowing cool oxygen across the face of the newborn. Heim (1971) stated that certain areas of the body play an important role in regulating heat production. The facial skin and mucous membranes of the upper respiratory tract have been determined to affect the thermoregulatory process. When cool oxygen is delivered to the face of an infant, a 23% increase in oxygen consumption occurs in full-term infants and 36% in premature infants. Any oxygen that is given to an infant should be warmed if this increase in oxygen consumption is to be avoided (Mestyán, Járai, Bata, & Fekete, 1964; Přibylova, 1968).

Premature Infants at Highest Risk to Lose Heat

A normal full-term infant is at risk to lose heat when compared to an adult, whereas a premature infant is at even greater risk due to his immaturity and physical characteristics. Providing a warm environment is even more important for the survival of the premature infant than for the full-term infant. Adamsons (1966) pointed out that a fall in body temperature of one to two degrees resulting from exposure to room temperature is unlikely to cause a full-term infant any serious problem and is generally well tolerated. Whereas, "sub-optimum conditions are more likely to affect the premature and the depressed infant due to their lower thermal stability" (Adamsons, 1966, p. 616).

Adamsons (1966) stated that tissues of low-metabolic activity can tolerate a wider range of temperature than can tissues of higher metabolic rate. Adamsons postulated whether or not this would apply to any given tissue at any level. If this is so, then the brain tissue of a premature infant should be less susceptible to cooling than that of a term infant since the metabolic rate of the brain in immature infants "must be substantially lower than that in the infant at term" (Adamsons, 1966, p. 600). This view is reflective of a minority group of researchers that have hypothesized that cooling of the infant actually decreases the need for oxygen.

In discussing premature infants and dysmature infants, Heim (1971) stated that thermal stability was diminished in these infants. In comparing the two groups, Heim found that the thermal stability in small-for-date babies was much better than for the premature infants of the same body size. This finding was in spite of the small-for-date babies' unfavorable body mass-surface area ratio and their lack of subcutaneous fat. Prematures are at greater risk for heat loss than small infants who are more mature. Sinclair (1975) pointed out that the smallest infants have the greatest difficulty maintaining body temperature in the delivery room. Smith (1969) also concurred and added that often the problem of maintenance of body temperature may be compounded with the interaction of hypoglycemia, metabolic acidosis, and respiratory distress syndrome. These conditions are all aggravated by cold exposure of the premature infant.

A major reason why premature infants are so susceptible to heat loss is their relatively large body surface area by which they can exchange heat. Korones (1972) stated that thermal stability is more precarious in infants weighing less than 2,000 gm. This is due largely to the ratio between body surface area and body weight.

> The larger the surface in relation to body mass, the greater the opportunity to dissipate heat to the environment. . . . The discrepancy between weight and body surface is even greater for low birth-weight infants. A larger surface area provides more extensive exposure to the environment, thus promoting more heat loss. This is the principle source of the neonate's difficulty with maintaining thermal stability. (p. 60)

Adamsons and Towell (1965) also agreed that thermoregulation in the premature infant is severely handicapped by the high surface area to body weight ratio. Brück (1961) stated that the heat loss in premature infants (1,500 gm) was greater than full-term infants. Brück estimated that this heat loss was at least 4.9 times as great as in adults per body surface unit, whereas full-term infants lose heat at four times the rate that adults lose heat per body surface unit. Philip (1977) stated that the premature infant has an exaggerated surface area to body weight ratio as a result of the premature's large head. This greater surface area intensifies the heat loss problem in the premature infant.

In addition to a high surface area, the premature is at risk to lose heat because of low tissue insulation. Premature infants characteristically have little body fat to serve as insulation from the environmental temperatures. Sinclair (1975) stated that it is the low thermal insulation of the newborn infant that limits the thermoregulatory range in which an infant can maintain his temperature. Oliver (1977) also credited the premature infant's decreased amount of body fat as being responsible for the limited ability to maintain body temperature observed in premature infants.

Closely related to the decreased amount of subcutaneous fat that serves as insulation for the infant is the premature infant's small body size. Adamsons (1966) found that thermal stability is a function of body size, surface area, thermal insulation, and the degree by which heat production can be increased to meet the demands of the environment. "It is evident that for a given body shape, thermal stability increases with increasing body size" (p. 600). A study by Hardman, Hey, and Hull (1969) showed that week-old rabbits achieve thermal stability over a much wider range of ambient temperatures than they were capable of on the day of birth. The researchers determined that this was due to the rabbits' larger size and increased insulation since a week-old rabbit has already doubled his birth weight.

Sinclair (1970) stated that the handicap of small body size is its limited thermal insulation. He described thermal insulation as consisting of two parts: (a) surface-air insulation and (b) tissue insulation. He found that small-for-date infants share with premature infants the thermoregulatory consequences imposed by small body size. "The smaller the body, the more easily does it exchange heat with the environment for a given gradient of temperature between skin and environment (low surface-air insulation)" (p. 151). Sinclair (1970) added that what was most important was the amount of tissue through which heat must be transferred. There is low tissue insulation in both premature and small-for-date infants.

Sinclair (1970) stated that the thermal handicap imposed by a small body size is not modified by the infant's gestational age but is a function of the small body size with its inherent deficiencies. Brück (1961) concurred with Sinclair that the thermal consequences imposed by a small body size are not modified by gestational age. Sinclair (1970), on the other hand, indicated that while the small-for-date infant shows the effects of small body size on his thermoregulatory range, he "may show comparatively well-developed homeothermic responses insofar as these depend on gestational age" (p. 148). Brück (1961) found that body size is related to basal metabolism. The basal metabolic rate increases with increasing body size; however, "basal metabolic rate cannot be described by a simple exponential function of weight . . . since in addition to body weight, age is also involved as a parameter in the function" (p. 73).

Another factor that increases the premature infant's rate of heat loss is posture. One of the characteristics of the premature is an extended posture when lying supine. The full-term infant, on the other hand, lies in a tightly flexed position. The more premature the infant is, the more extended are the extremities. Oliver (1977) said that heat loss is aggravated by the premature's posture which is due to neurologic immaturity and lack of flexor tone. Sinclair (1970) discussed how the infant's posture can increase or decrease the total surface area available for heat exchange.

> Babies have a relatively large surface area in relation to body mass. This surface-to-mass handicap is exaggerated in the infant of low birth weight. However, the surface available for heat loss by radiation and convection is not the total surface, but rather a portion of the total surface that varies with posture. Postures characteristic of various gestational ages have been described by Amiel-Tison. These postures tend toward

> increasing flexion of the extremities with increasing gestational age. Thus, the small-for-date infant achieves an effective surface area that is somewhat less than that of the preterm baby of similar size, and to this extent he can limit heat loss better. (p. 150)

Until about the last 25 years, the premature infant was thought to be poikilothermic, which means that he makes no attempt to regulate his body temperature when exposed to cold or hot temperatures. This concept was held longer for the premature than for the full-term infant. Adams et al. (1964) and Brück (1961) established that when a newborn experiences heat loss, he compensates by increasing his heat production. This reaction has also been observed in premature infants, "further disproving the hypothesis that homiothermy is a property acquired with increasing maturity after birth" (Adamsons, 1966, p. 608). Silverman and Agate (1964) supported the hypothesis that premature infants are homeothermic. These researchers found that babies under 1,500 gm who were exposed to mild cold stress did increase their heat production but were better able to increase their heat production if they were gestationally more mature. Silverman, Sinclair, and Agate (1966) had similar findings among infants less than 1,500 gm.

The thermal regulatory process begins just as quickly in premature infants as it does in full-term infants, but heat production in premature infants is unable to keep up with the amount of heat lost. Only when there is first, an increase in body size and an improvement in heat insulation and second, a greater depth of body and a thicker layer of fat does the thermal control range gradually expand. When premature infants are exposed to an environmental temperature of 23°C, they show an average 42% increase in their metabolic rate on the first day of life which increases to 90% during the first week (Brück, 1961).

Adamsons and Towell (1965) indicated that homeothermic responses are present in the premature. However, Adams et al. (1964) and Brück (1961) found that infants less than 30-32 weeks gestation do not increase their heat production when exposed to a cool environment. Stern, Lees, and Leduc (1965) demonstrated that some premature infants cannot initiate metabolic thermogenic processes as adequately as full-term infants. They attributed this inadequacy in producing heat to a decreased ability by premature infants to release noradrenalin, which has been postulated to be related to metabolism of brown fat.

Washington (1978) indicated that the premature infant has a diminished response to cooling.

> Being premature, he has a decreased amount of subcutaneous fat to act as insulation against the cold; a large skin surface area for his weight (which is all exposed to cold and from which heat can radiate); reduced amounts of "brown fat"; reduced caloric intake, which limits metabolic means of heat production; and decreased available oxygen resources,

> resulting from underdeveloped lungs that provide less oxygen to use in the metabolic thermogenic process. The effect of "cooling" can be potentially fatal. (p. 24)

In summary, a premature is capable of increasing his metabolism to increase his body temperature. However, some researchers point out that this increase many times is inadequate to counteract the large heat losses and is not as efficient as in full-term infants. Some prematures seem unable to increase their metabolism at all to any appreciable extent if they are very immature.

Consequences of Heat Loss In the Premature Infant

In considering the thermal conditions that are present at birth, at least in a typical hospital setting, "it remains to be elucidated whether the transient fall in peripheral and deep body temperature occurring immediately after birth favors or hinders adaptation to extrauterine life" (Adamsons & Towell, 1965, p. 538). Many researchers have suggested that the initial cold stimulus is influential in the onset of breathing. Adamsons and Towell (1965) postulated that cold may serve to increase the function of the reticular system. Likewise, cold induced vasoconstriction raises the systemic vascular resistance which will reduce the right to left shunt that is present in fetal circulation through the ductus arteriosus. Sinclair (1975) made reference to several studies that found that cooling plays "at least a contributory role in the resumption of breathing after birth" (p. 149). In a study by Harned and Ferreiro (1973) breathing was initiated after birth by various methods of cooling in full-term fetal lambs.

On the other hand, a warm environment has been documented to result in better survival rates for premature infants, beginning with Budin in the early 1900s and reconfirmed in the 1960s. Gandy et al. (1964) found that placing an infant under a radiant warmer to keep him warm results in a faster recovery from metabolic acidosis. Dahm and James (1972) stressed minimal cold stress by stating that prolonged cold stress may lead to hypoglycemia.

Perlstein, Edwards, and Sutherland (1970) observed that premature infants have an increased number of apneic attacks when the incubator temperature is rising. Besch et al. (1971), using the findings of Perlstein et al. (1970), stated that maintaining an infant's body temperature within a thermoneutral range avoids the risk of apneic attacks by having to warm a cool infant. While there is some controversy over whether a cool or warm environment is of greatest benefit to the newborn, most researchers have found that a warm environment provides greater benefits to the premature infant.

The ideal environment for a premature infant is the thermal neutral environment. This term refers to the environmental temperature in which oxygen consumption is minimal yet sufficient to maintain body temperature. The ideal ambient temperature for a full-term and premature infant is defined by Heim (1971) to be between 32-34°C with the air and inner-wall temperature of the incubator being equal and the relative humidity at 50%. Brück (1961) put the neutral ambient temperature at 32-33°C for full-term and premature infants. Brück (1961) stated that when there is the slightest drop in the ambient temperature below an infant's thermal neutral range the infant begins to lose his body temperature. This thermal neutral range expands with increasing body size.

There is not total agreement over what is the ideal skin temperature which reflects that the infant is in a thermal neutral environment. Heim (1971) suggested that an abdominal skin temperature of 36-37°C is the temperature in which oxygen consumption is minimal. Brück (1961) indicated that the normal range of core temperature is 36.5°C to 37.5°C. Silverman, Sinclair, and Agate (1966) observed that oxygen consumption was minimal when the abdominal skin temperature was 36°C. Hey and Katz (1970) put the temperature at 36.5°C. Silverman, Sinclair, and Scopes (1966) said that oxygen consumption is minimal at

36-37^{o}C skin temperature taken over the region of the liver. Ziegal and Van Blarcom (1972) stated that a skin temperature of 98^{o}F (36.7^{o}C) is necessary to prevent a metabolic and oxygen need increase. Brück (1961) correlated a drop in skin temperature with an increase in metabolism. Brück found that "in newborn infants, a lowering of the average skin temperature to about 35-36^{o}C (corresponding to an ambient temperature of 28^{o}C) is sufficient to produce and maintain a metabolic rise" (pp. 77-78).

One of the main effects of heat loss in a premature infant is an increase in metabolic rate in order to produce heat to maintain body temperature within a thermoneutral range. Hill (1959) stated that heat production in the premature infant is a chemical reaction leading to an increase in metabolic rate requiring that increased oxygen be available. Brück (1961) stated that when an infant is placed in an ambient temperature of 23^{o}C, the average temperature of the delivery room, the infant's metabolic rate increases 100%. If an adult is placed in this same environment, he shows either no metabolic rise or, at the most, an increase of 25%. Heat production in the infant at 23^{o}C is approximately the same as the maximum amount of heat production found in adults. Brück (1961) found that even when the infants were placed in an ambient temperature of 17.5^{o}C their heat production did not increase. Apparently maximum heat production occurs at $23^{o}C$ in infants. This fact gives evidence that the newborn is under more thermal stress under everyday conditions. Brück (1961) stated that the metabolic rise due to cold stress is greater than the increase due to either activity or ingestion of food. Sinclair (1975) further stated that when exposed to cold, the infant increases his metabolism by as much as 2-2½ times his resting rate which is "energetically an expensive one" (p. 147). This increase is present even at birth.

Another consequence of heat loss is increased oxygen need. This need is closely related to the above mentioned consequence of increased metabolic rate. Taylor (1971) stated that if an infant is reasonably well oxygenated "heat production, and therefore oxygen consumption, increases in response to a cool environment in an attempt to maintain body temperature" (p. 766). Silverman, Sinclair, and Agate (1966) found that oxygen consumption was related to a drop in skin temperature. Stephenson, Du, and Oliver (1970) did a study on the effect of a cool environment on arterial blood gas tensions in babies immediately after birth. Their results showed that the arterial pO_2 level was significantly lower in the cool group at 20 and 60 minutes of age but not at 4 hours. The pCO_2 was similar for both groups. The authors concluded that both a cool environment and asphyxia had similar effects on the infant.

Loss of body temperature in the premature infant results in a slower recovery from the metabolic acidosis that develops during the birth process. Gandy et al. (1964) designed a study to determine the effects of mild cold exposure on the rate of recovery from varying degrees of respiratory and metabolic acidosis at birth. They had two groups of infants: (a) a warm group whose body temperature was maintained at 37°C following birth by use of a radiant warmer and (b) a cool group whose temperature was allowed to fall by exposure to room temperature (25°C).

> Both groups showed a similar recovery of pH to normal levels following birth, but the "cool" group was only able to do so by increasing elimination of CO_2 to compensate for a developing metabolic acidosis. Infants depressed by birth asphyxia were unable to maintain pH during exposure to room temperature and developed a progressive metabolic acidosis. Thus, recovery from birth asphyxia can be seriously delayed by exposure to cold. (pp. 540-541)

The metabolic acidosis that may occur with cold stress has been associated with several acids. Schultz et al. (1979) demonstrated that hypothermia is associated with a high mean blood lactate level which falls with increasing rectal temperatures resulting in acidosis. They stated that their findings were in agreement with Gandy et al.

(1964). Gandy et al. (1964) named several acids as contributors to the acidosis.

> It is reasonable to suppose that the increased metabolism in a cool environment might lead to an accumulation of lactic, pyruvic and other organic acids, thereby aggravating the existing acidosis. This would be particularly true if metabolic needs were to exceed the oxygen available to the tissues. (p. 751)

Additionally, they postulated that a cool environment might aid in the acid-base homeostasis if the formation of these acid metabolites were decreased at a lower body temperature (Gandy et al., 1964).

Cold stress can aggravate or even cause an infant to become hypoglycemic. Cornblath and Schwartz (1976) found that hypoglycemia may occur in infants at four to six hours of age if the infant was chilled at birth. Chadd and Gray (1972) stated that hypoglycemia may be triggered by the increased metabolic rate that accompanies cold stress.

> This is true because glucose consumption is necessarily increased whenever there is an increase in metabolism. It has also be clinically demonstrated [by Stern, 1970] that when serum nonesterified fatty acid (NEFA) levels rise, as is the case when chemical thermogenesis is initiated, serum glucose levels fall. If the infant does become hypoglycemic, this complication may further contribute to acidosis as well as result in neurological damage if the hypoglycemia is not quickly corrected. (Williams & Lancaster, 1976, p. 358)

Heim (1971) mentioned that hypoglycemia is more frequent in premature and dysmature infants than in full-term infants. He hypothesized that perhaps the lower glucose level was responsible for the impaired thermal stability of this group. A group of premature infants and a group of dysmature infants were both subjected to a cold environment. In order to determine if a higher blood glucose level would result in am improved ability to produce heat, .5 - .75 g/Kg of glucose was infused over ten minutes. This resulted in a rise in the infant's blood sugar level. However, there was no effect on heat production in either group. While cold stress can cause an infant to become hypoglycemic, hypoglycemia is not responsible for the thermal instability of the premature infant.

Heat loss in premature infants has been found to interfere with their growth and weight gain. Williams and Lancaster (1976) stated that the increase in metabolism associated with cold stress leads to an increase in caloric consumption which would otherwise be used for cellular growth and weight gain. Glass et al. (1968) studied the rates of growth in low birth-weight babies that were exposed to different thermal environments between one and three weeks after birth. Each group received identical feedings of 120 calories per kilogram per day.

The babies in the thermoneutral environment grew faster in both weight and length than the cooler group. When the infants in the cooler group were given additional calories to make up for the cost of the increased metabolic rate incurred by the lower temperatures, the infants were able to catch up. Korones (1972) stated that weight gain in premature infants can be poor due to the partial diversion of the energy of metabolism to the production of body heat in preference to the laying down of new tissues. The fact that an infant is cared for in an unfavorable environment can interfere with his normal growth pattern.

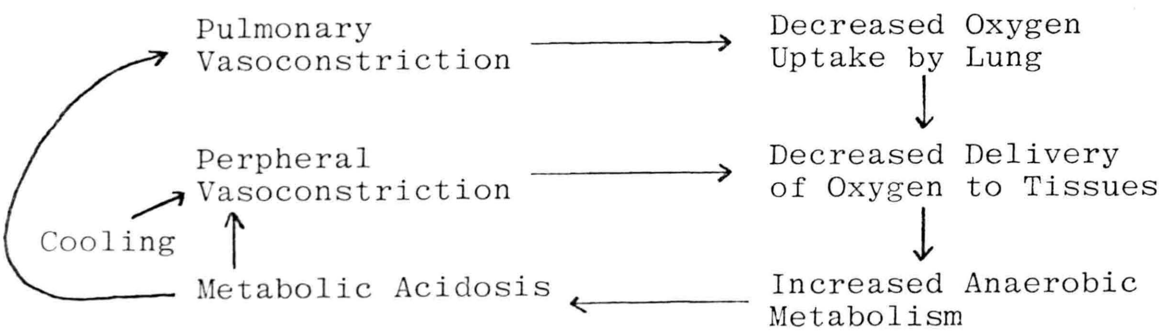
One of the protective mechanisms a premature infant has against heat loss is vasoconstriction. Oliver (1977) stated that peripheral vasoconstriction in response to cold is well developed at birth even in the premature infant. This vasoconstriction is aggravated by the metabolic acidosis occurring at birth and is prolonged by cold stress. This vasoconstriction can even extend to the pulmonary blood vessels.

> Vasoconstriction of pulmonary vessels, when severe, may lead to the development of three major problems: hypoxia, intracardiac shunting through the foramen ovale and aggravation of Respiratory Distress Syndrome Type I, if present. (Williams & Lancaster, 1976, pp. 357-358)

Stephenson et al. (1970) noted that cold stress can delay the establishment of the circulation to the postnatal pattern. The right-to-left shunting that is present in fetal circulation persists for a longer period due to vasoconstriction of the pulmonary vessels. Washington (1978), in discussing the phenomenon of vasoconstriction in response to cold, enumerated some of its effects along with other consequences of cold stress.

> Some complications of hypothermia are a noted increase in metabolic rate, which results in an increased respiratory rate per minute metabolic acidosis; persistent vasoconstriction; decreased tissue perfusion; and decreased pulmonary perfusion. There is also an increased norepinephrine and pulmonary resistance as well as hypoglycemia. (Washington, 1978, p. 26)

Philip (1977, p. 34) diagrammed the process:



One of the major complications of cold stress is its effect on the survival rate among infants, particularly premature infants. "The association of hypothermia with a diminished rate of survival in premature infants was suggested as early as 1900" (Korones, 1972, p. 58). Washington (1978) stated that "as neonatal nurses, we must be aware of the primary importance of thermal regulation of the neonate in sustaining his life" (p. 23). Besch et al. (1971) called a warm environment a "protective influence." Besch et al. (1971) speculated that since infants respond to a heat-losing environment by homeothermically increasing their metabolic rate, perhaps this added caloric expenditure contributes to infant mortality by depleting the infants' energy reserve necessary for preserving homeostatic functions. Sinclair (1970) underlined the lethal effect cold has on infants by saying that studies done by Buetow and Klein (1964), Day et al. (1964), and Silverman et al. (1958) demonstrated an increased mortality in the first days of life in low birth-weight infants who were raised in incubators set below the thermal neutral range. Sinclair (1970) stated,

> It is attractive to speculate that the energy cost of defending deep body temperature against cold contributes to the lethal effect (particularly in the first days of life, when the balance between caloric supply and caloric expenditure is marginal). (p. 155)

Related to the problem of increased mortality and cold stress, is the problem of maintenance of body temperature in infants being transferred between hospitals. Several referral centers have reported a relationship between survival of low birth-weight infants and their temperature on arrival at the center (Chance, O'Brien, &

Swyer, 1973). Therefore, a warm environment seems to be associated with an improved survival rate, especially among premature infants.

Three other factors have been associated with hypothermia. These are drug metabolism, kernicterus, and blood clotting problems. Sinclair (1975) stated that little research has been done on drug metabolism in newborns, but there have been numerous studies in animal research. A study by Morishima, Mueller-Heubach, and Shnider (1971) found that the half-life of lidocaine in newborn puppies increased from 58 to 83 minutes if the puppy was cooled. Applying this to the human newborn, one could reason that hypothermia would cause an infant to be slower to metabolize any kind of drug that might be in his system as a result of the mother receiving some medication during her labor.

Williams and Lancaster (1976) discussed the relationship of hypothermia with kernicterus and blood clotting. Stern (1970) found kernicterus to be related to hypothermia in the following manner:

> The increase in NEFA [nonesterified fatty acids] in the blood which is associated with thermogenic processes may lead to another problem--the development of kernicterus, even at relatively low levels of indirect bilirubin. Acidosis tends to cause a dissociation of indirect bilirubin from the major plasma protein responsible for binding with it--albumin. As long as bilirubin is bound

> to albumin, it cannot leave the vascular space and enter brain cells to cause damage. But NEFA also has a strong affinity for albumin, and as blood NEFA levels rise, particularly in the presence of acidosis, bilirubin-albumin binding may be replaced by NEFA-albumin binding. This will free indirect bilirubin in the blood circulation to travel to the brain parenchyma causing kernicterus. (Williams & Lancaster, 1976, p. 358)

Lastly, Williams and Lancaster (1976) suggested that severe hypothermia seems to interfere with certain clotting factors. They mentioned that other authors have noted an increase in pulmonary and cerebral hemorrhage in cold-stressed infants, however, this kind of bleeding is also associated with asphyxia.

In considering the effects of a cold environment on newborns, one of the more crucial areas is the effect of cold on a sick or depressed newborn. Many premature infants fall into these categories as many prematures have some kind of respiratory problems at birth due to their immature lungs. Washington (1978) stated that cold stress in an infant with a special medical problem can lead to a potentially fatal situation. Korones (1972) said that since cold stress results in an increase in the metabolic rate, the infant's oxygen consumption increases as well as his oxygen requirement.

> Thus a severely depressed infant can ill afford the oxygen cost imposed by cold stress at a time when he urgently needs the oxygen to maintain normal function of brain cells and cardiac muscle. (p. 59)

Taylor (1971) elaborated on this point by saying that the increased oxygen consumption experienced by the cold stressed newborn is made possible by an increase in his minute volume,

> and thus the newborn with respiratory distress may experience further difficulty when exposed to a cool environment and will, if unable to meet the demands for increased ventilation, become increasingly hypoxemic. (p. 766)

Gandy et al. (1964) did a study of the effects of the thermal environment on the acid-base status in infants. They found that babies placed in a cool environment show a rise in metabolic rate, a slower recovery from the acidosis of birth asphyxia, and hyperventilation. These researchers stated that healthy infants achieved a close-to-normal pH in the first 2 hours after birth, but depressed infants did not because they were unable to hyperventilate in response to cold and metabolic acidosis. This response occurred even if the infants were only depressed for a short period of time.

Ziegal and Van Blarcom (1972) stated that chilling of an infant interferes with the production of surfactant. For this reason, babies with hyaline membrane disease have a decrease in heat production and may be hypothermic even in relatively warm environments. Williams and Lancaster (1976) also mentioned the association Stern (1970) found between hypothermia and decreased production of surfactant. The authors restated Stern's findings that

> Hypoxia results in perfusion of oxygen which is precipitated by vasoconstriction. If perfusion of Type II alveolor cells is sufficiently inhibited in a newly born premature infant, the production of surfactant may be impaired, contributing to the development and/or severity of Respiratory Distress Syndrome. (Williams & Lancaster, 1976, p. 358)

Thus vasoconstriction which is induced by a cool environment can hinder the production of surfactant in premature infants.

One last area to be considered when thinking of thermal environment in depressed infants is the problem of cold stress during resuscitative procedures. Maintenance of body temperature is crucial in these infants. Washington (1978) stated that after five minutes of exposure of the infant during resuscitation, the infant may become so cold that his chances for revival diminish considerably. Maintaining an infant within his thermoneutral range is therefore particularly important to the depressed or critical infant. "In view of all the problems hypothermia may cause, preventing it rather than remedying it should be the goal" (Williams & Lancaster, 1976, p. 358).

Means of Maintaining Body Temperature

There are several means by which an infant's temperature can be maintained. Either the infant must regulate his own temperature or be assisted to maintain temperature within a thermoneutral range. When an infant is challenged by heat or cold, a normal baby will respond promptly to keep body temperature near its setpoint. An infant is capable of attempting to prevent heat loss by means of vasoconstriction and to maintain body temperature by increasing the rate of heat production (Sinclair, 1970). An infant is not totally helpless to defend his body temperature, neither is a premature infant. Adamsons and Towell (1965) stated that homeothermic responses are present in both the premature and the full-term infant. Vasodilation and vasoconstriction have been demonstrated to be present in premature infants along with an increased metabolic rate in response to low environmental temperatures (Adams et al., 1964; Brück, 1961). On the other hand, this response is absent or decreased in infants less than 30-32 weeks (Brück, 1961; Silverman & Agate, 1964). Berhman (1977) stated that the premature's vasoconstriction in response to a cool environment is quite adequate.

An infant's body temperature is dependent on two gradients, external and internal. Sinclair (1970) stated that the effect of vasoconstriction is to increase the internal (core-skin) temperature gradient and to increase tissue insulation to its maximum capacity. Williams and

Lancaster (1976) citing a study done by Burton related that:

> a decrease in body temperature is related to both the "internal gradient" (rectal/skin exchange) and "external gradient" (skin/air exchange). The neonate can exert some physiologic control over the internal gradient by vasoconstriction of blood vessels. But Hardy demonstrated that loss of heat by the external gradient is controlled solely by the environment. (p. 356)

Oh and Lind (1967) found that early clamping of the umbilical cord evokes a greater regional vasoconstriction in the infant which in turn improves the internal gradient.

> Skin vasoconstriction is the most powerful defense mechanism against heat loss, after early clamping of the umbilical cord, increased heat production remains the only mechanism to counter any further heat loss. (Heim, 1971, p. 793)

Vasoconstriction is one mechanism a premature infant has to safeguard body temperature.

Heat production is the second factor that the infant uses to keep body temperature from falling when exposed to environmental temperatures below the thermal neutral zone. Adamsons and Towell (1965) credited the newborn with the ability to increase metabolism in response to cold. This metabolic function is the mechanism by which balance is maintained between heat loss and heat production.

The process by which an infant produces heat is known as chemical thermoregulation. Chemical thermoregulation consists of two components: (a) shivering thermogenesis, which is heat production that is accompanied by electrical activity of the skeletal muscles and (b) non-shivering thermogenesis, which is heat production without visible or electric muscular activity (Heim, 1971). Non-shivering thermogenesis is a more effective and efficient form of chemical thermogenesis for the newborn (Brück, 1961). Sinclair (1970) credited the newborn with the ability to produce heat to maintain deep body termperature which can include muscular thermogenesis if the cold stress is sufficiently severe.

Brown fat or brown adipose tissue has been held to be responsible for heat production in newborns. This tissue is characterized by a rich blood and nerve supply, a high mitochondrial content, and a high metabolic rate in vitro. This tissue is found mainly in the intra-scapular region, axillae, perirenal areas, and around the large vessels in the thorax. There is some disagreement over whether this tissue is related to heat production. Dawkins and Hull (1964) found that a newborn piglet responds to exposure to cold by increasing the metabolic rate. This response is in spite of the fact that the newborn piglet has no adipose tissue either brown or white.

Most investigators do support the role of brown fat as being responsible for heat production. Hardman and Hull (1970) supported the contribution of brown fat to cold-induced thermogenesis. Their data showed that brown fat tissue continued to produce heat even after its own fat stores were depleted by the oxidation of glucose and fatty acids taken from the circulation. Aherne and Hull (1966) found that premature infants have a smaller amount of brown fat when compared to a mature infant. "It seems probable, therefore, that its capacity to produce heat is also less" (p. 232).

Some studies have been done that suggest that some part of brown fat metabolism is under the control of the sympathetic nervous system and that norepinephrine is the mediator in the process. Lees, Younger, and Babson (1966) noticed an increase in excretion of norepinephrine in infants who made successful homeothermic responses to environmental cold stress.

> Major attention has been directed recently to the role of cold and catacholamines in stimulating metabolism in brown adipose tissue, where catecholamine-induced enhancement of triglyceride breakdown appears responsible for thermogenesis. (Sinclair, 1970, p. 153)

If vasoconstriction and heat production are not sufficient to protect an infant's body temperature, the nurse must employ other means to prevent further heat loss. One such means is the use of an external source of heat. Incubators of many types are available today such as convection-heated incubators and radiant warmers. Results of a study by Miller and Oliver (1966) demonstrated that infants placed in a convection-heated incubator recovered their body temperature in a significantly shorter time than infants just wrapped in blankets after delivery. Likewise, Oliver (1965) found that infants placed in an incubator with the air temperature between 32-35^{o}C, after being dried, lost less body temperature. Even though the infants were placed in incubators, they still lost some body heat due to evaporative heat loss from wet hair and radiant heat loss to the cooler incubator walls.

Most modern delivery rooms are equipped with a radiant warmer which provides a source of external heat but still allows easy access and visibility of the infant. Du and Oliver (1969) stated:

> A radiant heater provides a suitably warm micro-environment for a baby in an air-conditioned delivery room. With this heater, temperature loss is minimized (if the baby is dried soon after birth), and at the same time, the infant remains exposed for close observation. (p. 1502)

A study was done by Rosen et al. (1978) to examine the thermoneutral conditions in the delivery room. All infants born vaginally were dried and placed under a radiant heater. All infants born by Cesarean section were dried and placed in a heated Isolette. The researchers did a retrospective chart review on the first three rectal temperatures taken on 135 newborns. The temperatures of the 108 infants born vaginally and the 27 infants delivered by Cesarean section were compared on admission and at four and eight hours after birth. The researchers found that the temperatures of all Cesarean section babies and 101 of the infants born vaginally were in the thermo-neutral range. There was also no difference found between the temperatures of infants born vaginally and infants delivered by Cesarean section. The researchers concluded that

> the present methods of thermoregulation in the delivery room seem satisfactory for infants with adequate brown fat deposits, but that infants who are small for dates or premature need special care to ensure heat preservation. Aware of their problems, delivery room staff can prepare better for the delivery of these infants. (p. 1693)

Another means of protecting the infant from heat loss is by increasing the insulation from the cool environment by means of clothing and wrapping the infant. Beland (1970) said that clothing the infant reduces heat loss because clothing is a poor conductor of heat and traps air which is also a poor conductor. "Because air is trapped in the spaces between fibers, loosely woven woolens are effective in preventing heat loss under certain conditions (when air is still)" (p. 596). Scopes and Ahmed (1966) reported that the temperature of the ambient air inside clothing is often as high as 35^{o}C even if the surrounding environmental temperature is 26^{o}C. Sinclair (1976) stated that the addition of clothing and bedding increased the infant's insulation to where the full-term infant can tolerate temperatures of 24°C and premature infants can tolerate 30°C. Clothing also helps to minimize the effects of sudden changes in environmental conditions.

An infant can lose heat through his scalp. In addition to being approximately 9-18% of the infant's total surface area, the head has less insulation than other parts of the body (Sinclair, 1976). Smith (1969) stated that "considerable heat loss can occur through the bare scalp of a small infant; consequently, the infant's head should be covered" (p. 653). Capobianco (1980) said that for premature infants "a tiny stocking cap can be beneficial, even inside the incubator. (A baby's head accounts for a large portion of his total body surface area)" (p. 67). Porth and Kaylor (1978) stated:

> One of the best means to prevent heat loss from conduction, convection, radiation, or evaporation is to supplement nature's thermoregulation by covering the infant and increasing the insulation. . . . Bonnets made to fit small infants prevent heat loss from the head after the infant leaves the incubator. (p. 1693)

"The head as a body surface contributing to heat loss is dramatized by the enhanced protection provided by swaddling of all but the exposed face of the infant" (Besch et al., 1971, p. 123). In their study, Besch et al. (1971) found that infants who had their heads covered retained more heat than those whose heads were bare. "This is not surprising since the head constitutes a considerable portion (9 to 18 percent) of the body surface of the newborn infant" (p. 123).

Closely related to clothing the infant to maintain body temperature, is the use of plastics to cover the infant. Smith (1969) mentioned the use of plastic drapes or silver foil to cover an infant as protection from the environment. Baum and Scopes (1968) developed a swaddler known as the silver swaddler that is made out of a polyester material that has been laminated on the inside with a thin nonabrasive layer of aluminum. Sinclair (1976) said that the silver swaddler has proven to be effective in preventing heat loss because it reflects all energy radiated from the baby's skin as well as preventing any kind of radiant heat loss. Valman (1979) mentioned the silver swaddler which was designed to limit heat loss during transport. He stated that the swaddler "is commercially available, but wrapping the baby with Gamgee wool is also satisfactory" (p. 1432). Besch et at. (1971) developed another swaddler made of a "unique swaddling material that proves to be at least as effective as the radiant heater in shielding against heat loss" (p. 123). This material is a plastic sheet with air pockets sealed between two layers of polyethylene coated with polyvinylidene. This material is used to cushion contents in commercial packages. One of the major hindrances of the silver swaddler is that the infant is hidden by the material. The proponents of the clear plastic swaddler say that the infant is more visible, but access to the infant is difficult for both the clear and foil-type swaddlers.

Skin Temperatures and Electric Thermometers

Several studies suggested that skin temperatures rather than rectal temperatures seem to be more closely related to the increased oxygen consumption that accompanies cold stress. Some researchers indicated that it is the difference in environmental temperature and skin temperature that determines the amount of metabolic rise. Miller and Oliver (1966) stated that

> recent studies provide further evidence that core (rectal) temperature is not the prime receptor site for thermal regulation. Brück, Silverman, and Agate and more recently, Adamsons and co-workers have shown this to be a function of skin receptor sites. (p. 968)

Silverman and Sinclair (1966) also mentioned that Brück and Day demonstrated that skin temperatures in newborns had an influence on the metabolic rate in newborn infants that appear unrelated to inner body temperature. Brück (1961) observed that oxygen uptake increased in the newborn immediately after experiencing external cooling. Since oxygen consumption increased so rapidly after being placed in a cool environment, this response must be triggered by cutaneous thermal receptors. Adamsons and Towell (1965) reported findings that suggested that the peripheral receptors that respond to differences in temperatures between the skin and the environment are more important in thermal regulation than are hypothalmic receptors. Adamsons, Gandy, and James (1965) reported that no correlation was found between oxygen consumption and rectal temperatures in healthy mature infants from birth to six hours old. Oxygen consumption varied by as much as 200% for a given rectal temperature. A closer relationship was demonstrated between oxygen consumption and skin or environmental temperatures. The best correlation was demonstrated when oxygen consumption was described as a function of the temperature gradient between body surface temperature and environmental temperature. "Thus, newborn infants have an increased sensitivity in regard to cutaneously induced metabolic rises" (Brück, 1961, p. 79).

There has been some disagreement over the amount of time required to obtain an accurate temperature. Eoff et al. (1974) did a study comparing axillary and rectal temperatures in newborn infants taken with a glass thermometer and a telethermometer. To carry out the study the authors reviewed recommendations found in the literature regarding the length of time a thermometer should be held in place to insure an accurate reading. They found that there was disagreement among authors over the amount of time necessary to obtain an accurate temperature. For axillary temperatures, time periods varying from $1\frac{1}{2}$ minutes to 10 minutes were recommended by Dunham (1961), Loudon (1957), and Nichols, Ruskin, Glor, and Kelly (1966). For rectal temperatures, a time period from 1 to 5 minutes was recommended by Fuerst and Wolf (1963), Metheny and Sniveley (1967), and Nichols and Glor (1968). Nichols et al. (1966) concluded from their study that oral temperatures are adequately measured in 7 minutes, axillary temperatures take 10 minutes and, rectal temperatures take 2 minutes.

Recently the electric thermometer has been developed to measure a person's temperature. Beck, Campbell, and St. Cyr (1977) stated that one problem noted with predictive electric thermometers is the report of inaccuracies due to the different temperatures which are in the mouth. "Except for this, electronic thermometers have a generally high performance in laboratory tests" (p. 28). Beck et al. (1977) also reported the concern of nurses over the accuracy of the thermometer as the battery wears down.

According to Gunthrie Laboratory, however, accuracy is maintained even after the recharge signal activates. IVAC Corporation (1979) reported that their thermometers are accurate within .1^{o}C and hold a charge for approximately 600 temperatures.

Summary

This chapter discussed thermoregulation as it affects the newborn infant, especially the premature infant. The chapter began with a brief overview of progress in thermoregulatory knowledge and techniques. Reasons why full-term infants as well as premature infants are susceptible to lose body heat was covered. The consequences of heat loss in infants were discussed. The means of maintaining body temperature were discussed from the role the infant plays to the techniques available to the nurse to use to maintain the infant's temperature. Various studies were presented correlating skin temperature to metabolic rate followed by information regarding different kinds of thermometers.

CHAPTER 3

PROCEDURE FOR COLLECTION AND TREATMENT OF DATA

This study utilized the classical experimental approach with a pre and post test design. In this design, there are two groups randomly selected from a given population and assigned to either the experimental group or the control group. A pre-test is given to both groups. The treatment is applied. Then both groups are given a post-test to determine the effect of the treatment (Treece & Treece, 1973). Polit and Hungler (1978) stated that a true experiment is characterized by three properties:

> 1. Manipulation--the experimenter does something to at least some of the subjects in the study.
> 2. Control--the experimenter introduces one or more controls over the experimental situation, including the use of a control or comparison group.
> 3. Randomization--the experimenter assigns subjects to a control or experimental group on a random basis. (p. 150)

In this study there was a population of premature infants that were randomly divided into a control group and an experimental group. The pre-test and post-test were the axillary temperatures obtained in the delivery room and nursery respectively. The treatment was the use of a stockinet cap.

Setting

This study took place in Parkland Memorial Hospital which provides general health services for a large metropolitan area of over 1,000,000 people in the Southwest. The hospital has 102 bassinets and 12 neonatal intensive care beds and delivers approximately one out of every three babies delivered in the area it serves. In 1979, 941 of the babies delivered were classified as low birth-weight. The neonatologist in charge of the nurseries estimated that approximately 600 of these infants were premature.

Physically, the labor and delivery suite is separated by several floors from the low birth-weight nursery and the intensive care nursery and is connected by an elevator designated for the purpose of transporting these infants to these nurseries. The regular newborn nursery is on the same floor as the delivery suite.

Population and Sample

The population for this study consisted of 40 infants between 32 and 36 weeks gestational age born at Parkland Memorial Hospital from seven o'clock in the morning until five o'clock in the evening Monday through Friday during the study period. The sample, which was the population, was divided into a control group and experimental group using the stratified random technique (Polit & Hungler, 1978).

At a time prior to the data collection, 60 index cards were utilized to pre-arrange randomization for the future population. Due to the expectation that infants would be dropped from the study because of their failure to fulfill all of the research criteria, 60 cards were used instead of the projected 40 cards. The total expected population was divided into infants who were 32-34 weeks gestational age and infants who were 35-36 weeks gestational age. Four cards were selected from the 32-34 week pile, two were designated control group and two were designated experimental group. The cards were numbered one through four and put in order according to the occurrence of the designated number in a table of random numbers. The 35-36 week pile was treated in like manner. Four more cards were selected from the 32-34 week pile with two designated as the control group and two designated as the experimental group. They were numbered five through eight and ordered according to the occurrence of their designated number in a table of random numbers. The 35-36 week pile received the same treatment. This was continued until all 60 cards had been processed. This method was chosen because of the expected small number of the youngest premature infants. The researcher desired to ensure that the smallest infants were equally divided into the control and experimental groups.

To be included in this study, an infant must have met several requirements. First of all, the gestational age as calculated from the mother's last menstrual period had to be verified by clinical evaluation. The clinical evaluation was the determining factor. The premature infant also had to have an appropriate weight for his age. His Apgar score must have been 7 or above at five minutes after birth. The infants could not have signs of any central nervous system defect such as seizures or hydrocephalus. Babies were delivered both vaginally and by Cesarean section. An infant was dropped from the study if he developed sepsis as determined by positive growth of pathogenic bacteria after the culture had undergone 7 days of incubation. No infant was included if the mother's temperature was 37.8°C or above during labor. Lastly, the infant had to be delivered in the delivery room.

Protection of Human Subjects

To insure that no human rights were violated by this study, permission was obtained from the Human Subject Review Committee of Texas Woman's University to conduct the study (see Appendix A). Permission was then obtained from the agency in which the study was done (see Appendix

B). Informed consent was obtained from the mother of the infant during early labor and prior to the mother's receiving any medication. If the mother was under 18 years of age and single, or mentally incapable of giving informed consent, permission was obtained from her legal guardian. The researcher explained to the mother or legal guardian the procedure for the study and the fact that there would be little or no risk to her infant. The researcher explained and expected benefits of this study as being (a) an improved body temperature of the infant on arrival in the nursery and (b) increased nursing knowledge concerning body temperature maintenance when planning care for a premature infant. To insure the anonymity of the subjects, the infants were designated by their band numbers which they received at birth multiplied by a constant known only to the researcher. The researcher offered to answer any question the mother had regarding the study. Each mother was assured that she could withdraw her permission for participation in the study at any time without loss of medical care to herself or her infant. Following the verbal explanation, the mother was given a written form to sign which explained the procedure for the study, the risk involved, and the expected benefit to the infant (see Appendix C).

Instruments

The instrument used to determine the axillary temperatures in the delivery room and the nursery was an IVAC electric thermometer Model 821 which gives the temperature in centigrade. This thermometer is calibrated at three temperature levels and is accurate within .1°C. The IVAC Corporation stated that 99% of the thermometers returned for service retained their accuracy after months and sometimes years of hospital use (IVAC, 1979). IVAC Corporation recommended that for axillary temperatures in newborns that the instrument be used in the test mode. This meant that: (a) the oral temperature probe was disconnected from the main unit, (b) the probe with its cover placed in the axilla, and (c) the infant's arm held next to his body for 5 minutes. After 5 minutes, the temperature probe was reconnected to the unit and the temperature was displayed (see Appendix D). The nursery axillary temperatures were obtained within 10 minutes after the infant arrived in the nursery. A standard glass wall thermometer was used to measure the temperature of the delivery room. All of the glass thermometers used in this study displayed the same temperature when placed in the same room for a period of a week.

The instrument used to determine the gestational age and weight classification of the infants in this study was "Newborn Maturity Rating and Classification." This tool, distributed by the Mead-Johnson Corporation, is used to classify infants according to age and weight. To determine an infant's age, one assigns a number for each of the categories listed on the tool that most closely resembles the infant under study. These numbers are added and the resulting score is correlated with a gestational age by refering to maturity rating scale. Next, the infant's length, head circumference and weight are plotted on the appropriate graphs using the estimated gestational age. If the length, head circumference and weight fall in the middle category then the infant is appropriate for gestational age (see Appendix E).

Two other instruments were used in this study. A stop watch was used to measure the five minute time period necessary for the axillary temperatures. The Subject Information Sheet was developed by the researcher for recording the data (see Appendix F). This data from this instrument was used to describe the sample.

Data Collection

The data was collected using the observation method. When a mother was expected to deliver an infant of 32-36 weeks gestation, permission was obtained prior to the administration of any medications. After determining that the mother was afebrile during labor, the researcher accompanied the mother to the delivery room for the birth of the baby. The temperature of the delivery room was recorded. The time of the infant's birth was used as the starting point for the times recorded in this study. The researcher dried the infant under a radiant warmer and then determined if the infant was in the control or experimental group by selecting the top index card in the corresponding gestational age category. Subsequently that card was discarded. If the infant was in the experimental group, he received a sterile stockinet cap and his axillary temperature was taken. If the infant was designated as being in the control group, only his axillary temperature was taken. The birth weight, sex, race, and 5 minute Apgar score of the infants in both groups was noted. The temperature of the transport incubator was obtained, if this mode was used to transfer the infant to the nursery. Within ten minutes after the infant had arrived in the nursery and had been placed in an incubator, the researcher obtained and recorded the axillary temperature for both groups. At a somewhat later time the infants' gestational age was verified by the researcher and his chart reviewed for central nervous system defects noted by the physician.

After 7 days, laboratory reports were checked for any positive blood cultures.

Treatment of the Data

The sample was described demographically as to sex, ethnic group, weight, Apgar score, gestational age, method of delivery, amount of time elapsed from birth to onset of drying, amount of time elapsed from birth to arrival in the nursery, temperature of the delivery room, temperature of the transport incubator, and the highest temperature of the mother during labor. The means were computed for both groups for all categories along with percentages and ranges for comparison purposes.

The mean and range of the axillary temperatures taken in the delivery room and in the nursery were computed along with the mean and range of the temperature difference between the delivery room and nursery axillary temperatures. The data was also analyzed in relation to the number of infants in both groups who had skin temperatures below the thermoneutral range on arrival in the nursery.

Hypothesis I and hypothesis II were tested by using the analysis of variance to determine if the application of a cap had a significant effect on (a) the admission nursery axillary temperature or (b) the amount of heat lost from the time the axillary temperature was taken in the delivery room until the axillary temperature was taken in the nursery. The significance level was set at .05.

Polit and Hungler (1978) have stated that analysis of variance is a parametric procedure useful in testing the significance of differences between two or several means. Analysis of variance breaks down the variability of a set of data into two components: "1) the variability resulting from the independent variable and 2) all other variability, such as individual differences, measurement unreliability, and so on" (p. 553).

Because of the occurrence of several factors beyond the direct control of the researcher, analysis of covariance was done to remove the effects of these factors on the dependent variables, the average skin temperature on arrival in the nursery and the difference in the delivery room axillary temperature and the nursery axillary temperature.

> The analysis of covariance tests the significance of differences between group means after first adjusting the scores of the dependent variable to eliminate the effects of the covariate . . . The variability in the dependent measure that can be explained by the covariate is removed from further consideration. Analysis of variance is performed on what remains of Y's variability to see whether, once the covariate is controlled, significant differences remain between the groups. (Polit & Hungler, 1978, p. 581)

The covariates in this study were: sex, ethnic group, method of delivery, amount of time elapsed from birth to drying, amount of time from birth to arrival in the nursery, temperature of the delivery room, temperature of the transport incubator, highest temperature of the mother, gestational age, and weight.

CHAPTER 4

ANALYSIS OF DATA

This chapter contains a quantitative description of the sample. The two hypotheses are presented and analyzed using analysis of variance to determine the significance of the differences in the group means. Additional findings are presented. Analysis of covariance was used to rule out the effects of selected covariates on the dependent variable. The chapter ends with a summary of the findings.

Description of Sample

The sample for this study contained 40 premature infants who were between 32 to 36 weeks gestational age, and who were appropriate for gestational age as determined by the newborn maturity rating and classification tool. These infants were born either vaginally or by Cesarean section in the delivery room. The oral temperatures of all mothers throughout their labors was below 37.8^{o}C. All infants included in the study had Apgar scores of 7 or above at 5 minutes after birth. All infants in the study failed to demonstrate any evidence of sepsis or any kind of central nervous system disorder.

These infants were assigned at birth to either the experimental group or the control group using the stratified random technique until a total of 40 infants was reached. There were 17 infants assigned to the experimental group and 23 infants assigned to the control group.

Demographically, the experimental group had four males and 13 females or 24% and 76% respectively. The control group had 12 males and 11 females or 52% and 48% respectively (see Table 1).

In the experimental group there were six white infants (35%), five black infants (29%), and six Mexican-American infants (35%). The control group had eight white infants (35%), 13 black infants (57%), and two Mexican-American infants (9%) (see Table 1).

These infants were born either vaginally or by Cesarean section. The experimental group had 15 infants or 88% that were delivered vaginally and two infants or 12% delivered by Cesarean section. The control group had 18 infants or 78% who were born vaginally and five infants or 22% that were delivered by Cesarean section (see Table 1).

Infants in both groups ranged in gestational age from 32 to 36 weeks. The experimental group consisted of five infants in the 32-34 week age bracket or 29% and 12 infants in the 35-36 weeks age bracket or 71%. The control group had 4 infants in the 32-34 week age bracket or 17% and 19 infants in the 35-36 weeks age bracket or 83%. The mean gestational age in the experimental group was 34.9 weeks whereas the mean age in the control group was 35.2 weeks (see Table 1).

Table 1

Demographic Characteristics of the Experimental and Control Groups

Demographic category	Experimental[a] *f*	%	Control[b] *f*	%
Sex				
Male	4	24	12	52
Female	13	76	11	48
Ethnic group				
White	6	35	8	35
Black	5	29	13	57
Mexican-American	6	35	2	9
Method of Delivery				
Vaginal	15	88	18	78
Cesarean Section	2	12	5	22
Gestational Age				
32 to 34 weeks	5	29	4	17
35 to 36 weeks	12	71	19	83

[a]*n* = 17.
[b]*n* = 23.

These infants ranged in weight from 1,360 gm to 2,965 gm. The experimental group ranged from 1,420 gm to 2,890 gm with a mean weight of 2,253.4 gm. The range of the control group was from 1,360 gm to 2,965 gm with 2,276.8 gm as the mean weight.

The Apgar scores at 5 minutes after birth ranged from 7 to 9 for all infants. In the experimental group, 16 infants received a score of 9 or 94% with the remaining infant receiving a score of 7. The control group had 22 infants who received a score of 9 or 96%. The other infant received an Apgar score of 8.

There were two time periods that were measured in this study. The first was the amount of time that elapsed from the time of birth to the beginning of the drying procedure. The second was the amount of time that elapsed from the time of birth to arrival in the nursery. The average time involved from birth to the onset of drying was 1.9 minutes in the experimental group with a range of 1 to 5 minutes. The control group ranged from 1 to 9 minutes with an average time of 2.7 minutes. The average amount of time from birth to arrival in the nursery for the experimental group was 24.9 minutes with a range of 15 to 42 minutes. The control group ranged from 9 minutes to 52 minutes with an average time of 23.7 minutes.

It is the policy of Parkland Memorial Hospital that all infants under 2,500 gm go to the low birth-weight nursery on the seventh floor and be transported by a transport incubator. All infants weighing more than 2,500 gm go to the regular newborn nursery on the same floor as the delivery room and are carried there wrapped in a blanket. Due to this policy, some infants in both groups were transported to their nurseries in incubators and some were hand-carried. In the experimental group, 7 infants or 41% were transported in an incubator. Twelve infants or 52% of the control group were transported in an incubator to the low birth-weight nursery. The average temperature of the transport incubator for the experimental group was 35.3°C with a range of 31°C to 40°C. The control group had a mean transport incubator temperature of 35.2°C with a range from 33°C to 37°C. The remainder of the infants, 10 infants or 59% for the experimental group and 11 infants or 48% for the control group, were carried to the regular newborn nursery.

The temperature of the delivery room was recorded at the time of delivery for all infants. The average temperature of the delivery room was 21.9°C in the experimental group and ranged from 20°C to 23°C. The control group had a delivery room temperature range from 20°C to 22°C with an average temperature of 21.5°C.

The temperature of the mother was taken every 4 hours during labor. The highest temperature during labor was recorded. The average highest temperature recorded for the mothers of the experimental group was 37.1°C with a range of 36.6°C to 37.7°C. The average highest temperature recorded for the mothers of the control group was 37.2°C with a range of 36.2°C to 37.8°C.

Axillary temperatures were obtained from all infants in the delivery room as soon as the infant was dried. The average axillary temperature taken in the delivery room was 36.4°C for the experimental group with a range of 35°C to 37.4°C. Two infants (12%) in the experimental group had axillary temperatures below 36°C. The control group had an average delivery room axillary temperature of 36.2°C with a range of 35.3°C to 37.3°C. Seven infants (30%) in the control group had axillary temperatures below 36°C.

The axillary temperature was again taken on all infants upon their arrival in the nursery after they had been placed in their incubators. The average nursery axillary temperature recorded for the experimental group was 36.0°C with a range of 35.2°C to 36.9°C. Eight infants in the experimental group or 47% had an axillary temperature below 36°C. The control group had an average nursery axillary temperature of 35.9°C with a range of

34.8°C to 36.8°C. Twelve infants in the control group, or 52%, had axillary temperatures below 36°C.

The average amount of heat lost from birth to arrival in the nursery was determined by the temperature difference between the delivery room axillary temperature and the nursery axillary temperature. The average temperature difference for both groups was .3°C. The experimental group had a range of -1.1°C to .3°C and the control group had a range of -1.1°C to .6°C.

Findings

Analysis of variance was used to test hypothesis one: there will be no difference on arrival in the nursery in the admission axillary temperatures of premature infants who receive a stockinet cap after delivery and premature infants who do not receive a cap after delivery. The average axillary temperature on arrival in the nursery for those infants who received a cap was 36.0°C. The average axillary temperature on arrival in the nursery for those infants who did not receive a cap was 35.9°C. Analysis of variance, using the .05 level of significance, showed that there was no significant difference in the average nursery axillary temperatures of premature infants who received a cap and premature infants who did not receive a cap after delivery, $\underline{F}$ (1,38) = .56, $\underline{p}$ = .46.

Therefore, hypothesis one predicting no difference was accepted as stated.

Analysis of variance was also used to test hypothesis two: there will be no difference in the amount of heat lost from the time the axillary temperature is obtained in the delivery room until the axillary temperature is obtained in the nursery in premature infants who receive a stockinet cap after delivery and premature infants who do not receive a cap after delivery. The average amount of heat lost for both the experimental and control groups was .3°C. The results of analysis of variance, using the .05 level of significance, showed that there was no significant difference in the amount of heat lost in premature infants who received a cap and premature infants who did not receive a cap after delivery, $\underline{F}$ (1,38) = .03, $\underline{p}$ = .87. Therefore, hypothesis two predicting no difference was accepted as stated.

Additional Findings

Since temperature of an infant can be affected by several factors, the effect of these factors was statistically removed to determine if a difference in the two groups could be demonstrated. This was done by using analysis of covariance. The covariates considered were: sex, ethnic group, weight, method of delivery, amount of time elapsed from birth to arrival in the nursery, temperature of the delivery room, temperature of the transport incubator, delivery room axillary temperature, gestational age, and highest temperature of the mother. The .05 level of significance was used to determine whether the covariates had a significant effect.

The covariates that had the greatest effect on the nursery axillary temperature were: delivery room axillary temperature, sex, temperature of the mother, method of delivery, and weight. The delivery room axillary temperature had a significant effect, F (1,33) = 46.37, p = .001. The effect of sex was not significant, F (1,33) = 2.31, p = .14. The effect of the mother's temperature was not significant, F (1,33) = .07, p = .79. The method of delivery had a significant effect, F (1,33) = 5.34, p = .03. Weight did not have a significant effect, F (1,33) = .01, p = .94. The analysis of covariance showed that when both groups were statistically equalized for all five covariates, there was still no significant difference found in the adjusted means. F (1,33) = .12, p = .73 (see Table 2).

Analysis of covariance was also used to determine if a cap had a significant effect on the amount of heat lost from the time the axillary temperature was obtained in the delivery room until the axillary temperature was obtained in the nursery. This heat loss was measured as the

Table 2

Summary Table of Results of Analysis of Covariance Showing Effects of the Five Most Important Covariates in Relation to Nursery Axillary Temperatures

Source of variation	df	F	p
Covariates			
Delivery Room Axillary Temperature	1	46.37	.001**
Sex	1	2.31	.14
Mother's Temperature	1	.07	.79
Method of Delivery	1	5.34	.03*
Weight	1	.01	.94
Main Effects			
Group	1	.12	.73
Residual Error	33		
Total	39		

*$p < .05$.
**$p < .001$.

temperature difference between the delivery room axillary temperature and the nursery axillary temperature. The .05 level of significance was used to determine if the covariates had a significant effect.

The covariates that were found to have the most effect on this temperature difference were: sex, ethnic group, method of delivery, temperature of the transport incubator, and gestational age. The effect of sex was not significant, F (1,33) = .70, p = .41. The ethnic group had a significant effect, F (1,33) = 4.69, p = .04. The method of delivery also had a significant effect, F (1,33) = 5.47, p = .03. The incubator temperature did not have a signifant effect, F (1,33) = .80, p = .38. The effect of gestational age was not significant, F (1,33) = 2.85, p = .10. The analysis of covariance showed that when both groups were statistically equalized for these five covariates the remaining difference between the group means was not significant at the .05 level, F (1,33) = .10, p = .75 (see Table 3).

Since thermal stability is more precarious in infants weighing less than 2,000 gm, the researcher compared the difference in the nursery axillary temperatures of the experimental infants and the control infants under 2,000 gm using the Mann-Whitney U test. There were six infants in the experimental group and four infants in the control group who weighed less than 2,000 gm. The mean nursery axillary temperature in the experimental group was 36.1°C and ranged from 35.4°C to 36.9°C. The mean nursery axillary temperature for the control group was 35.4°C and ranged from 34.8°C to 36.2°C. The .05 level of significance was again used to determine if the cap had a

Table 3

Summary Table of Results of Analysis of Covariance Utilizing the Five Most Important Covariates on the Amount of Heat Lost

Source of variation	df	F	p
Covariates			
Sex	1	.70	.41
Ethnic Group	1	4.69	.04*
Method of Delivery	1	5.47	.03*
Incubator Temperature	1	.80	.38
Gestational Age	1	2.85	.10
Main Effects			
Group	1	.10	.75
Residual	33		
Total	39		

*$p < .05$.

significant effect. The result of the analysis showed that premature infants less than 2,000 gm who received a cap after delivery had significantly higher axillary temperatures on arrival in the nursery, $U = 3.00$, $p = .03$.

The mean delivery room axillary temperatures of the two groups were different. The mean delivery room axillary temperature for the experimental group was 36.4°C with a range of 36.1°C to 37.1°C. The mean delivery room axillary temperature for the control group was 35.6°C with a range of 35.3°C to 36.3°C. Analysis of covariance was done to determine if the higher nursery axillary temperatures observed in the experimental group was a result of the cap or their higher delivery room axillary temperatures. The analysis showed that the delivery room axillary temperature had a significant effect on the nursery axillary temperature, F (1,7) = 25.78, p = .001. When the two groups were statistically equalized in regard to their delivery room axillary temperatures, no significant difference between the two groups could be demonstrated, F (1,7) = .16, p = .70 (see Table 4). Therefore, a stockinet cap had no effect on the nursery axillary temperatures once the difference in the delivery room axillary temperatures was controlled in premature infants weighing less than 2,000 gm.

The effect of a cap on the amount of heat lost from the time the axillary temperature was obtained in the delivery room until the axillary temperature was obtained in the nursery was also examined for the 10 infants who weighed less than 2,000 gm. Both groups lost the same amount of heat, .3°C. The experimental group ranged from a loss of .9°C to a gain of .1°C. The control group ranged from no loss of temperature to a loss of .7°C. The results of the Mann-Whitney U showed that there was no significant

Table 4

Results of Analysis of Covariance Determining the Effect of a Cap on Nursery Axillary Temperature Controlling for Delivery Room Axillary Temperature in Infants Weighing Less Than 2,000 gm

Source of variation	df	F	p
Covariate			
Delivery Room Axillary Temperature	1	25.78	.001*
Main Effects			
Group	1	.16	.70
Residual Error	7		
Total	9		

*$p < .001$.

difference between the two groups $U = 9.5$, $p = .31$. Therefore, a cap had no effect on the amount of heat lost in premature infants weighing less than 2,000 gm.

Summary of Findings

Analysis of variance was used to test the two hypotheses. The result of this analysis showed that a stockinet cap had no effect on a premature infant's temperature on arrival in the nursery. The results also showed that a cap had no effect on the amount of heat lost by a premature infant from the time the axillary temperature was obtained in the delivery room until the axillary temperature was obtained in the nursery.

Because body temperature can be influenced by several factors, analysis of covariance was done to remove the effect of these factors. The five most important covariates that had the greatest influence on an infant's nursery axillary temperature were: delivery axillary temperature, method of delivery, sex, temperature of the mother, and weight. Delivery room axillary temperature and method of delivery had a significant effect on the nursery axillary temperature. With both groups equalized in regard to the five most important covariates, there was still no difference in the nursery axillary temperature.

The five most important covariates to affect the amount of heat lost from the time the axillary temperature was obtained in the delivery room until the axillary temperature was obtained in the nursery were: the method of delivery, ethnic group, gestational age, temperature of the transport incubator, and sex. The method of delivery and ethnic group were the only two covariates which had a significant effect on this temperature loss. With both groups equalized in regard to all five covariates, there was still no significant difference found in the amount of heat lost in premature infants who received a cap and premature infants who did not receive a cap after delivery.

An additional finding showed that infants weighing less than 2,000 gm who received a stockinet cap after delivery had a higher admission nursery axillary temperature than premature infants who did not receive a cap after delivery. This difference was nullified when the effect of the delivery room axillary temperature was removed from the nursery axillary temperatures by analysis of covariance. The two groups also did not differ in the amount of heat lost from the time the axillary temperature was obtained in the delivery room until the axillary temperature was obtained in the nursery.

CHAPTER 5

SUMMARY OF THE STUDY

This study was carried out to determine whether or not the use of a stockinet cap would aid in maintaining a premature infant's body temperature resulting in primary prevention of cold stress. This study tested two hypotheses: (a) there will be no difference on arrival in the nursery in the admission axillary temperatures of premature infants who receive a stockinet cap and premature infants who did not receive a cap after delivery, and (b) there will be no difference in the amount of heat lost from the time the axillary temperature is obtained in the delivery room until the axillary temperature is obtained in the nursery in premature infants who receive a stockinet cap after delivery and premature infants who do not receive a cap after delivery.

Summary

A group of 40 premature infants who met all the research criteria were assigned to the experimental or control group by the stratified random technique. The experimental group received a stockinet cap after delivery as soon as possible after being dried under a radiant warmer. The axillary temperature was taken in the delivery room and again in the nursery. The control group received the same treatment minus the cap. Analysis of variance was used to determine if the cap had an effect upon the nursery axillary temperatures or the amount of heat lost by premature infants from the time their temperature was obtained in the delivery room to their arrival in the nursery. Analysis of covariance was also used to rule out the effect of various factors affecting the infants' axillary temperatures.

The results of these analyses showed that the use of a stockinet cap had no effect on a premature infant's nursery axillary temperature or the amount of heat lost from the time the axillary temperature was obtained in the delivery room until the axillary temperature was obtained in the nursery. When the data were analyzed using only those infants weighing less than 2,000 gm, a stockinet cap appeared to have a significant effect on the nursery axillary temperatures, but this effect was determined to be due to initial differences between the two groups and not the result of the cap. The cap had no effect either on the amount of heat lost from the time the axillary temperature was obtained in the delivery room until the axillary temperature was obtained in the nursery in premature infants weighing less than 2,000 gm.

Results of analysis of covariance showed that two covariates had a significant effect on nursery axillary temperature. The delivery room axillary temperature had the greatest effect on the nursery axillary temperature and was significant at the .001 level. The method of delivery also had a significant effect with $p = .03$. Two covariates were also found to have an effect on the amount of heat lost from the time the axillary temperature was obtained in the delivery room until the axillary temperature was obtained in the nursery. The method of delivery had the greatest effect with $p = .03$. Ethnic group also had a significant effect with $p = .04$.

Discussion of Findings

A study by Besch et al. (1971) looked at the effect of a transparent baby bag on rectal temperatures in newborn infants. These researchers found that the addition of a head-covering to these bags resulted in lower temperature falls in infants both under radiant heaters and those in room temperature. This study differed from the present study in that the head covering involved was a plastic material and that rectal temperatures were used to measure the effect of the bag. The results of the present study using a stockinet cap on premature infants did not show this lower temperature fall reported by Besch et al. (1971).

Porth and Kaylor (1978) reported that bonnets help prevent heat loss in small infants when they are taken out of their incubators. Capobianco (1980) also mentioned a benefit in using a "tiny stocking cap" on premature infants while they are still in their incubators. The results of the present study failed to demonstrate a benefit due to using a stockinet cap in preventing heat loss in premature infants after delivery.

Data was collected regarding several factors that can influence an infant's skin temperature. Gestational age was mentioned by Brück (1961), Heim (1971), and Silverman, Sinclair, and Agate (1966) as affecting body temperature regulation. Sinclair (1970) stated that gestational age does not modify a small infant's response to cooler temperatures; instead, the response is mainly a function of body size. The results of the present study supported Sinclair's findings in 1970 that gestational age has little effect on the amount of heat lost in the period following delivery.

Rosen et al. (1978), in their study comparing temperatures of infants born vaginally and by Cesarean section, found no difference in the rectal temperatures of these two groups. The results of the present study showed that the method of delivery had an effect on the nursery axillary temperature and the amount of heat lost from the time the axillary temperature was obtained in the delivery room until the axillary temperature was obtained in the nursery. This could have been due to the fact that axillary temperatures were used rather than rectal temperatures in the present study. Also the temperature of the radiant warmer may have been warmer for infants delivered by Cesarean section than for infants delivered vaginally due to the fact that the bed was heated for a longer period of time because the time of delivery could be more accurately predicted than for vaginal deliveries. The treatment of Cesarean section infants also varied in that these infants were not held by their mothers as were those infants of mothers who delivered vaginally. Rosen et al. (1978) combined the rectal temperatures on admission to the nursery, at 4 hours, and 8 hours after birth to arrive at a mean rectal temperature to use in comparing the two groups. Perhaps if the present study included the temperatures at 4 and 8 hours after birth, the results would be similar.

One of the goals of nursing care of a newborn infant is to prevent cold stress by maintaining an infant in a thermoneutral environment. Several researchers have determined that this environment is evidenced by a skin temperature between 36-37°C (Brück, 1961; Heim, 1971; Hey & Katz, 1970; Silverman, Sinclair & Agate, 1966; Silverman, Sinclair, & Scopes, 1966). The results of the present study showed that 20 infants out of the total 40 infants had axillary temperatures below 36°C on arrival in the nursery and that the infants lost little heat after the initial axillary temperature was obtained in the delivery until the axillary temperature was obtained in the nursery. Therefore, these infants were not maintained in a thermoneutral environment in the delivery room and experienced some degree of cold stress. Rosen et al. (1978) stated that the present method of providng a thermoneutral environment in the delivery room is adequate for full-term infants but small-for-dates or premature infants require "special care to ensure heat preservation" (p. 1693). The number of babies with axillary temperatures below 36°C in the present study has underlined the need for a renewed effort in the delivery room to increase thermal protection for premature infants.

Conclusions and Implications

The conclusions that can be drawn from this study were:

1. Additional use of a stockinet cap on premature infants does not aid in primary prevention of cold stress.

2. The gestational age of the infant has little effect on amount of heat lost after delivery.

Implications for nursing practice are:

1. Nurses receiving a premature infant at delivery should place the most emphasis on preventing heat loss in the initial minutes after birth if their goal of primary prevention of cold stress is to be achieved.

2. Nurses should investigate additional or alternate methods of preventing heat loss in premature infants after delivery to ensure maintenance of thermoneutral conditions.

Recommendations for Further Study

Based on the findings of this study, the following suggestions are submitted for future studies.

1. A study should be done with a larger sample, using a thermometer that can register temperature faster and display the readings continuously. A method for recording the radiant warmer temperature should be incorporated.

2. This study should be replicated using only infants under 2,000 gm.

3. A study should be done using different materials for a cap such as a plastic or aluminum-lined plastic.

4. A study should be done comparing the temperature of infants delivered vaginally and those delivered by Cesarean section to see if there is a difference due to the method of delivery or if the difference observed in the present study was the result of other factors.

5. This study should be replicated utilizing both rectal and axillary temperatures.

APPENDIX A

TEXAS WOMAN'S UNIVERSITY
Box 23717, TWU Station
Denton, Texas 76204

1810 Inwood Road
Dallas Inwood Campus

HUMAN SUBJECTS REVIEW COMMITTEE

Name of Investigator: Jenifer Roberts   Center: Dallas

Address: 6600 Turtle Creek   Date: 6/20/80

Dallas, Texas 75205

Dear Ms. Roberts:

Your study entitled Preventing Cold Stress in Premature Infants by Use of a Stockinet Cap

has been reviewed by a committee of the Human Subjects Review Committee and it appears to meet our requirements in regard to protection of the individual's rights.

Please be reminded that both the University and the Department of Health, Education, and Welfare regulations typically require that signatures indicating informed consent be obtained from all human subjects in your studies. These are to be filed with the Human Subjects Review Committee. Any exception to this requirement is noted below. Furthermore, according to DHEW regulations, another review by the Committee is required if your project changes.

Any special provisions pertaining to your study are noted below:

_____ Add to informed consent form: No medical service or compensation is provided to subjects by the University as a result of injury from participation in research.

_____ Add to informed consent form: I UNDERSTAND THAT THE RETURN OF MY QUESTIONNAIRE CONSTITUTES MY INFORMED CONSENT TO ACT AS A SUBJECT IN THIS RESEARCH.

_____The filing of signatures of subjects with the Human Subjects Review Committee is not required.

_XX_ Other: 1. Note that anonymity is further protected if you report the data as group data rather than report any individual scores. *

_____No special provisions apply.

Sincerely,

Estelle D. Kurtz

Chairman, Human Subjects Review Committee

at Dallas

* 2. The hospital I.D. number for the infant has the possibility of being identifying data. We suggest that you further code the number by multiploying or dividing the number by a constant known only by you.

PK/smu/3/7/80

APPENDIX B

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING

AGENCY PERMISSION FOR CONDUCTING STUDY*

THE Parkland Memorial Hospital

GRANTS TO Jenifer Roberts, R.N.

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

The problem of this study will be to determine if the use of a stockinet cap will affect heat loss in premature infants from birth until admission in the nursery.

The conditions mutually agreed upon are as follows:

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

Date: 7-23-80

[illegible signature]
Signature of Agency Personnel

Jenifer Roberts
Signature of Student

Judith A. Erlen
Signature of Faculty Advisor

*Fill out & sign three copies to be distributed as follows: Original - Student; First copy - Agency; Second copy - TWU College of Nursing.

APPENDIX C

TEXAS WOMAN'S UNIVERSITY
COLLEGE OF NURSING

Consent to Act as a Subject for Research and Investigation:

The following information is to be read to or read by the subject. One copy of this form, signed and witnessed, must be given to each subject. A second copy must be retained by the investigator for filing with the Chairman of the Human Subjects Review Committee. A third copy may be made for the investigator's files.

1. I hereby authorize Jenifer Roberts, R.N., a graduate student at Texas Woman's University or her assistants to perform the following procedures:

to be present at my delivery, to put a sterile stockinet cap on my infant, to measure his temperature, accompany him to the nursery, and again take his temperature. I understand that the investigator will be collecting information about my temperature in labor, the weight, sex, race, condition of my infant at birth, his age in weeks, and any health problems my infant may have.

2. The procedure or investigation listed in Paragraph 1 has been explained to me by Jenifer Roberts, R.N.

3. (a) I understand that the procedures or investigations described in Paragraph 1 involve the following possible risks or discomforts:

I understand that the procedure discussed in Paragraph 1 involves minimal or no risk to my infant. If the cap interferes with any kind of treatment, I have been assured that the cap will be removed immediately in order to perform the treatment and my infant will be withdrawn from the study. I have been assured that my identity will be protected. I understand that my infant will be identified by his band number that he receives at birth multiplied by a constant known only to the investigator and her assistants.

3. (b) I understand that the procedures and investigations described in Paragraph 1 have the following potential benefits to myself and/or others:

an improved body temperature of my infant on arrival in the nursery. In addition, the results of the study may give some direction for nurses in maintaining body temperature in premature infants.

3. (c) I understand that no medical service or compensation is provided to the subjects by the university as a result of injury from participation in research.

4. An offer to answer all of my questions regarding the study has been made. If alternative procedures are more advantageous to me, they have been explained. I understand that I may terminate my participation in the study at any time.

__________________________ __________________________
Subject's Signature Date

If the subject is a minor, or otherwise unable to sign, complete the following:

Subject is a minor, age_____, or is unable to sign because:

Signatures - one required

__________________________ __________________________
Father Date

__________________________ __________________________
Mother Date

__________________________ __________________________
Guardian Date

__________________________ __________________________
Witness - one required Date

APPENDIX D

Axillary Temperature Procedure

Regarding the recommended method for taking axillary temperatures, especially for the neonatal patient, our present recommendations are to use the thermometer in the "test" mode. This is accomplished as follows:

1. Remove the blue oral probe from the storage well in the thermometer and then unplug the connector from the front of the thermometer.

2. Apply an IVAC Model P950 cover to the probe.

3. Insert the probe in the axilla, making sure the tip of the probe is positioned as close to the axillary artery as possible. Keeping the probe pointed generally at the axillary artery and keeping the patient's arm close to his side, should allow the probe to remain in place unassisted.

4. Leave the probe in place for approximately four to five minutes then without moving the probe plug the probe connector into the front of the Model 811 Thermometer. The thermometer will automatically be energized in the "test" mode and will indicate accurately the patient's axillary temperature.

5. Record the temperature in the normal manner, remove the probe, discard the cover, and insert the probe back in the thermometer storage well.

IVAC Corporation, San Diego, California

APPENDIX E

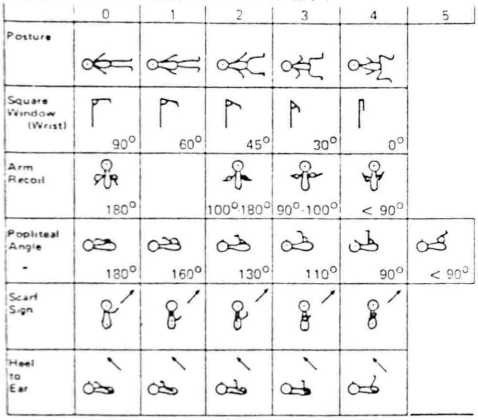
NEWBORN MATURITY RATING
and
CLASSIFICATION

ESTIMATION OF GESTATIONAL AGE BY MATURITY RATING

Side 1

Symbols: X - 1st Exam O - 2nd Exam

NEUROMUSCULAR MATURITY



	0	1	2	3	4	5
Posture						
Square Window (Wrist)	90°	60°	45°	30°	0°	
Arm Recoil	180°		100°-180°	90°-100°	< 90°	
Popliteal Angle	180°	160°	130°	110°	90°	< 90°
Scarf Sign						
Heel to Ear						

PHYSICAL MATURITY

	0	1	2	3	4	5
SKIN	gelatinous red, trans-parent	smooth pink, visible veins	superficial peeling &/or rash, few veins	cracking pale area, rare veins	parchment, deep cracking, no vessels	leathery, cracked, wrinkled
LANUGO	none	abundant	thinning	bald areas	mostly bald	
PLANTAR CREASES	no crease	faint red marks	anterior transverse crease only	creases ant. 2/3	creases cover entire sole	
BREAST	barely percept.	flat areola, no bud	stippled areola, 1–2 mm bud	raised areola, 3–4 mm bud	full areola, 5–10 mm bud	
EAR	pinna flat, stays folded	sl. curved pinna, soft with slow recoil	well-curv. pinna, soft but ready recoil	formed & firm with instant recoil	thick cartilage, ear stiff	
GENITALS Male	scrotum empty, no rugae		testes descend-ing, few rugae	testes down, good rugae	testes pendulous, deep rugae	
GENITALS Female	prominent clitoris & labia minora		majora & minora equally prominent	majora large, minora small	clitoris & minora completely covered	

Scoring system: Ballard JL, *et al*: A Simplified Assessment of Gestational Age, Pediatr Res 11:374, 1977. Figures adapted from "Classification of the Low Birth Weight Infant" by AY Sweet in Care of the High-Risk Infant by MH Klaus and AA Fanaroff, WB Saunders Co, Philadelphia, 1977, p 47.

Gestation by Dates ____________ wks

Birth Date ________ Hour ________ am/pm

APGAR ________ 1 min ________ 5 min

MATURITY RATING

Score	Wks
5	26
10	28
15	30
20	32
25	34
30	36
35	38
40	40
45	42
50	44

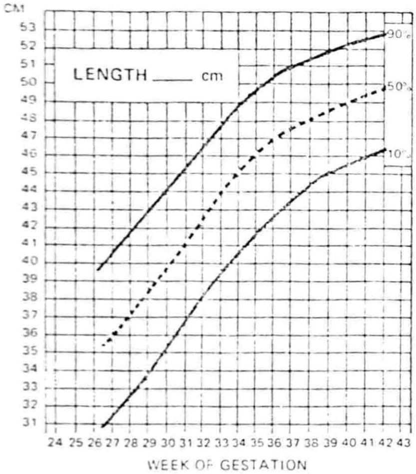
SCORING SECTION

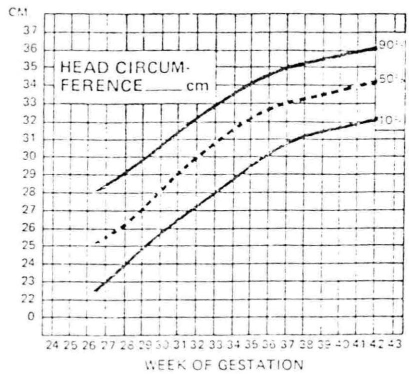
	1st Exam=X	2nd Exam=O
Estimating Gest Age by Maturity Rating	______ Weeks	______ Weeks
Time of Exam	Date ______ Hour ______ am/pm	Date ______ Hour ______ am/pm
Age at Exam	______ Hours	______ Hours
Signature of Examiner	______ M.D.	______ M.D.

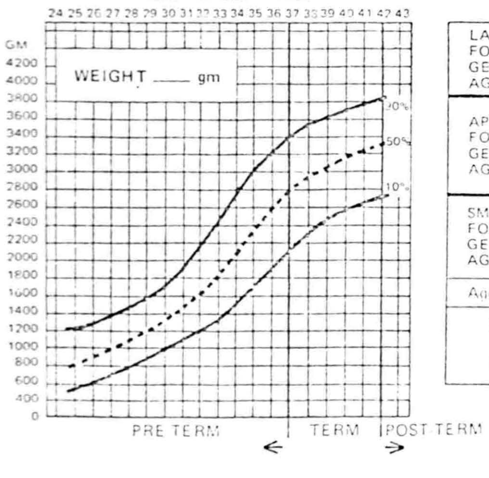
Side 2

CLASSIFICATION OF NEWBORNS – BASED ON MATURITY AND INTRAUTERINE GROWTH

Symbols: X - 1st Exam O - 2nd Exam







	1st Exam (X)	2nd Exam (O)
LARGE FOR GESTATIONAL AGE (LGA)		
APPROPRIATE FOR GESTATIONAL AGE (AGA)		
SMALL FOR GESTATIONAL AGE (SGA)		
Age at Exam	hrs	hrs
Signature of Examiner	M.D.	M.D.

Lubchenco LC, Hansman C, and Boyd E: Pediatr 37:403, 1966; Battaglia FC, and Lubchenco LC: J Pediatr 71:159, 1967.

APPENDIX F

SUBJECT INFORMATION SHEET

Subject___(code no.)_ Group__________ Gestational Age_______

Sex_______ Ethnic Group__________ Apgar Score in 5 minutes___

Weight of infant______________ Method of delivery___________

Highest temperature of mother during labor__________________

Time of:

Birth____________________________

Procedure begun_________________

Arrival in Nursery_____________

Temperature of:

Delivery Room_______________

Incubator____________________

Axillary Temperatures:

Delivery Room___________________ Nursery______________________

Verified Gestational Age______________________________________

Appropriate for Gestational Age - Yes_________ No________

Any Central Nervous System Defects or Problems______________

Blood Culture After 7 Days: Positive______ Negative______

REFERENCE CITATIONS

Adams, F. H., Fujiwara, T., Spears, R., & Hodgman, J. Temperature regulation in premature infants. Pediatrics, 1964, 33, 487-495.

Adamsons, Jr., K. The role of thermal factors in fetal and neonatal life. Pediatric Clinics of North America, 1966, 13, 599-619.

Adamsons, Jr., K., Gandy, G. M., & James, L. S. The influence of thermal factors upon oxygen consumption of the newborn human infant. Journal of Pediatrics, 1965, 66, 495-508.

Adamsons, Jr., K., & Towell, M. Thermal homeostasis in the fetus and newborn. Anesthesiology, 1965, 26, 531-548.

Aherne, W., & Hull, D. Brown adipose tissue and heat production in the newborn infant. Journal of Pathology and Bacteriology, 1966, 91, 223-234.

Baum, J. D., & Scopes, J. The silver swaddler. Lancet, 1968, 1, 672-673.

Beck, W. C., Campbell, R., & St. Cyr, B. Comparing thermometers. Nursing Care, 1977, 10(4), 14-15, 28-29.

Beland, I. L. Clinical nursing. Patho-physiological & psychosocial approaches (2nd ed.). New York: Macmillan, 1970.

Besch, N. J., Perlstein, P. H., Edward, N. K., Keenan, W. J., & Sutherland, J. M. The transparent baby bag--a shield against heat loss. New England Journal of Medicine, 1971, 284, 121-124.

Brück, K. Temperature regulation in the newborn infant. Biologia Neonatorum, 1961, 3(2/3), 65-119.

Buetow, K. C., & Klein, S. W. Effect of maintenance of "normal" skin temperature on survival of infants of low birth weight. Pediatrics, 1964, 34, 163-170.

Burnard, E. D., & Cross, K. W. Rectal temperature in the newborn after birth asphyxia. British Medical Journal, 1958, 2, 1197-1199.

Capobianco, J. A. Keeping the newborn warm: How to safeguard the infant against life-threatening heat loss. Nursing 80, 1980, 10, 64-67.

Chadd, M. A., & Gray, O. P. Hypothermia and coagulation defects in the newborn. Archives of Disease in Childhood, 1972, 47, 819-821.

Chance, G. W., O'Brien, M. J., & Swyer, P. R. Transportation of sick neonates, 1972: an unsatisfactory aspect of medical care. Canadian Medical Associates Journal, 1973, 109, 847-851.

Cornblath, M., & Schwartz, R. Disorders of the carbohydrate metabolism in infancy. (2nd Ed.) Philadelphia: W. B. Saunders, 1976.

Dahm, L. S. & James, L. S. Newborn temperature and calculated heat loss in the delivery room. Pediatrics, 1972, 49, 504-513.

Dawkins, M. J., & Hull, D. Brown adipose tissue and the response of new-born rabbits to cold. Journal of Physiology (London), 1964, 172, 216-238.

Day, R. L., Caliguiri, L., Kamenski, C., & Ehrlich, F. Body temperature and survival of premature infants. Pediatrics, 1964, 34, 171-181.

Du, J. N., & Oliver, Jr., T. K. The baby in the delivery room-a suitable microenvironment. Journal of American Medical Association, 1969, 207, 1502-1504.

Dunham, E. C. Premature infants (3rd Ed.). New York: Harper & Row, 1961.

Eoff, M. F., Meier, R. S., & Miller, C. Temperature measurements in infants. Nursing Research, 1974, 23, 457-460.

Fuerst, E. V., & Wolf, L. Fundamentals of nursing (4th ed.). Philadelphia: J. B. Lippincott, 1963.

Gandy, G. M., Adamsons, Jr., K., Cunningham, N., Silverman, W. A., & James, L. S. Thermal environment and acid-base homeostasis in human infants during the first few hours of life. Journal of Clinical Investigation, 1964, 43, 751-758.

Glass, L., Silverman, W. A., & Sinclair, J. C. Effect of the thermal environment on cold resistance and growth of small infants after the first week of life. Pediatrics, 1968, 41, 1033.

Göpfert, H. & Stufler, R. Die vorstadien des kaltezitterns bei geringer abkühlung des menschen. Pflügers Arch. ges. Physiol., 1952, 256, 161.

Hardman, M. J., Hey, E. N., & Hull, D. The effect of prolonged cold exposure on heat production in new-born rabbits. Journal of Physiology (London), 1969, 205, 39-50.

Hardman, M. J., & Hull, D. Fat metabolism in brown adipose tissue in vivo. Journal of Physiology (London), 1970, 206, 263-273.

Harned, H. S., Jr., & Ferreiro, J. Initiation of breathing by cold stimulation: Effects of change in ambient temperature on respiratory activity of the full-term fetal lamb. Journal of Pediatrics, 1973, 83, 663-669.

Heim, T. Thermogenesis in the newborn infant. Clinical Obstetrics and Gynecology, 1971, 14, 790-820.

Heim, T., & Hull, C. The blood flow and oxygen consumption of brown adipose tissue in the new-born rabbit. Journal of Physiology (London), 1966, 186, 42-55.

Hey, E. N. & Katz, G. The optimal thermal environment for naked babies. Archives of Diseases in Childhood, 1970, 45, 328-334.

Hill, J. R. Oxygen consumption of newborn and adult mammals: its dependence on the oxygenation in the inspired air and on the environmental temperature. Journal of Physiology (London), 1959, 149, 346-373.

IVAC Corporation. "Acceptable" isn't good enough for you. San Diego, California, May 1979.

Klaus, M. H., & Fanaroff, A. A. Care of the high-risk neonate. Philadelphia: W. B. Saunders, 1973.

Korones, S. B. High-risk newborn infants: The basis for intensive care nursing. St. Louis: C. V. Mosby, 1972.

Lees, M. H., Younger, E. W., & Babson, S. G. Thermal requirements of undergrown human neonates. Biologia Neonatorum, 1966, 10, 288-302.

Loudon, I. S. On taking temperature in the mouth and the axilla. Lancet, 1957, 57, 233-235.

Mestyán, J., Járai, I., Bata, G., & Fekete, M. The significance of facial skin temperature in the chemical heat regulation of premature infants. Biologia Neonatorum, 1964, 7, 243-254.

Metheny, N. M., & Sniveley, Jr., W. D. Nurses' handbook of fluid balance. Philadelphia: J. B. Lippincott, 1967.

Miller, D. L., & Oliver Jr., T. K. Body temperature in the immediate neonatal period: The effect of reducing thermal losses. American Journal of Obstetrics and Gynecology, 1966, 94, 964-969.

Morishima, H. O., Mueller-Heubach, E., & Shnider, S. M. Body temperature and disappearance of lidocaine in newborn puppies. Anesthesia and Analgesia, 1971, 50, 938-942.

Neuman, B. The Betty Neuman health-care systems model: A total person approach to patient problems. In J. P. Riehl & Sister C. Roy (Eds.), Conceptual models for nursing practice. New York: Appleton-Century-Crofts, 1974, 99-110.

Nichols, G. A., & Glor, B. A. Replication of rectal thermometer placement studies. Nursing Research, 1968, 17, 360-361.

Nichols, G. A.. Ruskin, M. M., Glor, B. A., & Kelly, W. H. Oral, axillary, and rectal temperature determinations and relationships. Nursing Research, 1966, 15, 307-310.

North, W. C. Control of the body temperature in the pediatric patient. North Carolina Medical Journal, 1961, 22, 59-63.

Oh, W., & Lind, J. Body temperature of the newborn infant in relation to placental transfusion. Acta Paediatrica Scandinavica, 1967, 172 (supplement), 137-145.

Oliver Jr., T. K. Temperature regulation and heat production in the newborn. Pediatric Clinics of North America, 1965, 12, 765-779.

Oliver Jr., T. K. Thermal Regulation. In R. E. Behrman (Ed.), Neonatal-perinatal medicine: Diseases of the fetus and newborn (2nd ed.). St. Louis: C. V. Mosby, 1977.

Perlstein, P. H., Edward, N. K., & Sutherland, J. M. Apnea in premature infants and incubator-air-temperature changes. New England Journal of Medicine, 1970, 282, 461-466.

Philip, A. G. Neonatology, a practical guide. Flushing, N.Y.: Medical Examination Publishing, 1977.

Polit, D. F., & Hungler, B. P. Nursing research: Principles and methods. New York: J. P. Lippincott, 1978.

Porth, C. M., & Kaylor, L. E. Temperature regulation in the newborn. American Journal of Nursing, 1978, 78, 1691-1693.

Přivylova, H. The importance of thermoreceptive regions for the chemical thermoregulation of the newborn. Biologia Neonatorum, 1968, 12, 13-22.

Rosen, E., Dupuis, H., & Wichman, N. Comparison of temperatures of neonates born vaginally and by C-Section. American Journal of Nursing, 1978, 78, 1693.

Schultz, K., Soltész, G., Molnár, D., & Mestyán, J. The effect of hypothermia on plasma metabolites in preterm newborn infants with particular references to plasma free amino acids. Biology of the Neonate, 1979, 36, 220-224.

Scopes, J. W., & Ahmed, I. Range of critical temperatures in sick and premature newborn babies. Archives of Diseases in Childhood, 1966, 41, 417-419.

Selye, H. The stress of life. New York: McGraw-Hill, 1956.

Silverman, W. A., & Agate Jr., F. J. Variation in cold resistance amoung small newborn infants. Biologia Neonatorum, 1964, 6, 113-127.

Silverman, W. A., Fertig, J. W., & Berger, A. P. The influence of the thermal environment upon the survival of newly born premature infants. Pediatrics, 1958, 22, 876-885.

Silverman, W. A., & Sinclair, J. C. Temperature regulation in the newborn infant. New England Journal of Medicine, 1966, 274, 146-147.

Silverman, W. A., Sinclair, J. C., & Agate Jr., F. J. Oxygen cost of minor variations in heat balance of small newborn infants. Acta Paediatrica Scandinavica, 1966, 55(3), 294-300.

Silverman, W. A., Sinclair, J. C., & Scopes, J. W. Regulation of body temperature in pediatric surgery. Journal of Pediatrics, 1966, 1, 321-329.

Sinclair, J. C. Heat production and the thermoregulation in the small-for-date infant. Pediatric Clinics of North America, 1970, 17, 147-158.

Sinclair, J. C. Metabolic rate and body size of the newborn. Clinical Obstetrics and Gynecology, 1971, 14, 840-854.

Sinclair, J. C. The effect of the thermal environment on neonatal mortality and morbidity. In K. Adamsons Jr., and H. A. Fox (Eds.), Preventability of perinatal injury (Vol. 2), New York: Alan R. Liss, 1975.

Sinclair, J. C. Metabolic rate and temperature control. In C. A. Smith, & N. M. Nelson (Eds.), The physiology of the newborn infant (4th ed.). Springfield, Illinois: Charles C. Thomas, 1976.

Smith, R. M. Temperature monitoring and regulation. Pediatric Clinics of North America, 1969, 15, 643-659.

Stephenson, J. M., Du, J. N., & Oliver Jr., T. K. The effect of cooling on blood gas tensions in newborn infants. Journal of Pediatrics, 1970, 76, 848-852.

Stern, L. The newborn infant and his thermal environment. Current Problems in Pediatrics, 1970, 1, 2-29.

Stern, L., Lees, M. H., & Leduc, J. Environmental temperature, oxygen consumption, and catecholamine execretion in newborn infants. Pediatrics, 1965, 36, 367-373.

Taylor, P. M. Respiratory distress in the newborn. Clinical Obstetrics and Gynecology, 1971, 14, 763-789.

Thauer, R., & Wezler, K. Der stoffwechsel im dienste der wärmeregulation (erste und zweite chemische wärmeregulation). Z. ges. exp. Med., 1943, 112, 95.

Treece, E. W., & Treece, J. W., Jr. Elements of research in nursing. St. Louis: C. V. Mosby, 1973.

Valman, H. B. Infants of low birth weight. British Medical Journal, 1979, 2, 1431-1432.

Washington, S. Temperature control of the neonate. Nursing Clinics of North America, 1978, 13(1), 23-28.

Williams, J. K., & Lancaster, J. Thermoregulation of the newborn. Maternal Child Nursing, 1976, 1, 355-360.

Ziegal, E., & Van Blarcom, C. C. Obstetric nursing (6th ed.). New York: Macmillan, 1972.