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ROUTINE CARE OF THE NORMAL NEONATE

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The importance of compulsive routine care of the newborn is often overlooked because the basic physiology and pathology underlying this care has been forgotten. This article serves to refresh the memory on the physiology of hyperbilirubinemia, hemorrhagic disease of the newborn, and gonococcal eye prophylaxis, among other topics. With new understanding, the approach to the low-risk neonate is seen as having an important rationale.

The recent literature has an abundance of articles concerning the recognition and care of the low-birth-weight or high-risk neonate. This emphasis occurred as a result of increased awareness of the problems of this group of newborns who require a disproportionate amount of time and who represent those infants most at risk of dying. Often forgotten in this flurry of advancing technology are the term, or low-risk, infants. Important to their remaining in this low-risk category is careful nursing attention to ensure that three general aspects of newborn care are met: the detection of significant medical problems early, protection from environmental and internal processes to which they are susceptible, and promotion of good health by aiding in the adaptation to extrauterine life.

DETECTION OF SIGNIFICANT MEDICAL PROBLEMS

The process of detecting significant medical problems begins in the labor suite where the antepartum nurse monitors the labor. This is done in many hospitals with electronic fetal heart rate monitoring, and the nurse is often the first one to recognize a problem. This may be late decelerations, which indicate fetal hypoxemia from a maternal, placental, or fetal event that limits oxygenation of the fetus; or variable decelerations that result from umbilical-cord compression. As these may indicate fetal distress, the physician is notified so that intervention may occur.[1] This role is continued in the delivery room,

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where the obstetrical nurse may be the only person available to recognize the need for resuscitation or to recognize a major congenital abnormality. She or he also participates in the transition to extrauterine life by manipulating the environment to minimize stress. Newborn infants have just left an environment where temperature was not a factor and entered a delivery suite that may be drafty and cold. They are wet, so heat loss occurs by evaporation, convection, and conduction. The consequences of hypothermia include respiratory distress, metabolic acidosis, and delayed resuscitation. The nurse can minimize hypothermia by drying babies with a warm towel and placing them under a radiant warmer or near another adequate heat source.[2]

At this point, many babies are assigned to Level II care for surgical problems, prematurity, birth injury, or asphyxia while others are assigned to Level III care for respiratory support. The remainder go to Level I care, or a traditional low-risk nursery, where the low-risk nurse will continue to select out infants with possible medical problems. The first consideration is to determine whether infants arriving in the transitional area are in fact low-risk infants. The nurse should give each infant a brief physical examination including checking vital signs (temperature, respiratory rate, heart rate, and blood pressure), observing respiratory distress (grunting, flaring, retracting, tachypnea, or cyanosis), palpating the abdomen, and auscultating the heart. If there are no obvious abnormalities he or she must then determine whether the infant is full term. The World Health Organization uses 37 weeks as the dividing line for term infants, but there is some evidence that infants at 37 weeks may still have increased morbidity and mortality.[3] The designation of term birth as occurring at 38–42 weeks gestation is more general, for it includes 80 percent of all deliveries and it is this group of infants who have the lowest neonatal mortality. Thus, infants of less than 38 weeks gestation are preterm; those born at 38–42 weeks gestation are term; and those born at greater than 42 weeks are postterm. Gestational age is determined from two sources, (1) the estimate based on the mother's last menstrual period and (2) a clinical examination of the baby. The clinical assessment is the most accurate; each infant's gestation should be assessed by one of the standard techniques. These assessments were originally done by two different methods, either by the use of external characteristics or by neurological examination.[4,5] Most forms in use now are a combination of the two, such as the clinical assessment of gestational age developed by Dubowitz, Dubowitz, and Goldberg, which allows accurate determination of gestational age within 2 weeks.[6]

This assessment, in addition to giving a raw score for gestational age and an idea of risk, provides a point on the abscissa of the chart to determine the status of the birth weight. In previous classifications infants weighing less than or equal to 2500 g were considered preterm, whereas those weighing greater than 2500 g were considered full term. Pioneers in the field of neonatology demonstrated that this was not true,[7] so that in addition to the concern about gestational age there then developed the concept of appropriate weight for gestational age. Using one of the standard growth curves such as the Colorado Intrauterine Growth Curve,[8] infants can now be placed in one of nine categories: preterm appropriate for gestational age (PrAGA), small for gestational age (PrSGA), or large for gestational age (PrLGA); full-term appropriate for gestational age (FAGA), small for gestational age (FSGA), or large for gestational age (FLGA); and postterm appropriate for gestational age (PoAGA), small for gestational age (PoSGA), or large for gestational age (PoLGA). It is the continuing care of the full-term, appropriate-for-gestational-age (FAGA) infants that is of concern here.

PROTECTION FROM ENVIRONMENTAL AND INTERNAL PROCESSES

Infection

FAGA infants have been found by a nursing physical examination and by assessment of gestational age to be at low risk for any immediate medical problems. The next goal is to protect them from a group of illnesses or abnormalities to which they are susceptible by virtue of their age. First and foremost comes protection from infection because they are uniquely susceptible. Full-term neonates stand a significant risk of 1 in 1200 of becoming infected and if infected a greater than 50 percent chance of dying.[9] This is because they do not have an intact immunologic system. Protection begins at the body surface, where nonspecific host defenses, especially the skin, prevent invasion by bacteria. The skin of newborns is often not intact because of abrasions from forceps, punctures from fetal scalp electrodes, and skin breakdown from trauma. They also have surgical sites, the umbilicus and the circumcision; and these two areas of devitalized tissue are readily colonized by pathogenic organisms. Once bacteria have breached the local defense mechanisms, the immunologic defenses are called into play. The primary, or nonspecific, immune response is the inflammatory response, which requires that while blood cells (polymorphonuclear cells) be able to

migrate to the site of invasion, attach to the organisms, ingest the organisms, and destroy them. The cells of newborn infants have been demonstrated to have diminished ability to migrate,[10] as well as decreased killing power,[11] thus their primary immune response is inadequate.

The secondary, or specific, immune defense is the production of specific antibodies, which is important primarily in augmenting white blood cell function. Specific antibodies attach to organisms and thus prepare them for phagocytosis or ingestion by the white cells. The two most important classes of antibodies are immunoglobulin G (IgG), which provides immunity to the majority of organisms, and immunoglobulin M (IgM), which is particularly important in gram-negative infections. As fetuses have not usually been exposed to infection *in utero*, their antibody levels come from transplacental passage of maternal antibodies reflecting their mothers' past exposure to infection. Although IgG levels may be high, newborns may still be susceptible to certain organisms to which their mothers had not been exposed. In addition, IgM is not transferred across the placenta, so newborns are therefore at increased risk for gram-negative infection.[12] In view of inadequate protection at all levels of the immunologic system it is surprising that infection does not occur more often.

Some of the newborn infections such as the one caused by the β-hemolytic streptococci of group B are not preventable by nursery practices because they are probably acquired during birth.[13] Other infections can be prevented. The first preventive measure occurs in the delivery room with Credé's method for preventing gonococcal ophthalmia neonatorum. The importance of this care can be emphasized by looking at the history of this disease. From 1906 to 1911, 24 percent of children admitted to U.S. schools for the blind were disabled as a result of gonococcal ophthalmia neonatorum. During 1958–1959 only 0.3 percent of the children at these schools were sightless because of gonococci.[14] With the rising incidence of venereal disease, however, the incidence will increase with inadequate prophylaxis. At present, all 50 states require some form of prophylaxis, and at least 22 of them specify silver nitrate, which is the prophylaxis recommended by the American Academy of Pediatrics.[15] There have been failures of prophylaxis, but these are probably due to failure of technique. No topical drug can work if it fails to come into contact with the organism. To have the optimal effect this drug must be placed in the conjunctival sac. The eye must be opened for instillation, and it should not be rinsed out with either saline solution or water as this may diminish its effectiveness.

The eye care is usually done immediately after birth, but it can be delayed for 30 minutes so as not to interfere with eye-to-eye contact between mother and infant.

The next preventive measure is to minimize the opportunity for infants to be colonized by pathogenic organisms. This is facilitated by appropriate attire in the nursery. Those who are in the nursery continuously should wear scrub dresses. In nurseries where babies are cared for in bassinets or open warmers, the personnel who are not regularly in the nursery should wear gowns tied in the back to cover front and back of clothing. Gowns are important, but they may give a false sense of security. They must not diminish the importance of the most successful barrier to infection, which is good hand washing. Most infectious agents responsible for disease in the nursery are transmitted from infant to infant by the hands of nursery personnel. To minimize this possibility before entering the nursery, all should wash hands and arms to the elbows with an antiseptic agent after removing jewelry.

The two most popular antiseptic agents are hexachlorophene and iodide preparations. The major disadvantage of hexachlorophene is that it is active against gram-positive organisms only. *Escherichia coli*, a gram-negative organism, can be cultured from the hands of nurses after a scrub with hexachlorophene. Everyone is aware of the fact we do not use hexachlorophene on babies because of evidence of neurotoxicity, but there is some concern from Sweden, which has linked birth defects to repeated hand washing with hexachlorophene soap. In a review of 460 births to nurses, there was a 15 percent incidence of birth defects in infants born to nurses who used hexachlorophene routinely, compared with a 3.4 percent incidence in a comparison group of nurses not using hexachlorophene. The incidence of birth defects in the general population is 3 percent. This study has been criticized, and as a result the Center for Disease Control has recommended further studies before taking an official stand on the use of hexachlorophene by pregnant women. As there are alternatives, others are suggesting that pregnant women not use hexachlorophene.[16] Iodophor preparations are probably the most suitable because of their effectiveness against both gram-positive and gram-negative organisms. Their major problem is that they cause skin sensitization.[17] A more recently introduced product, chlorhexidine gluconate, is considered by some to be an improvement over the other two preparations as it is effective against both gram-positive and gram-negative organisms and has minimal potential for skin sensitization.[18]

The organism that causes most problems postnatally is *Staphylo-*

coccus aureus, which readily colonizes the skin, umbilical cord, nares, and circumcision site in the first week of life. It has been shown that fewer than 10 bacteria will initiate umbilical colonization in the majority of neonates. The disease can occur as generalized sepsis, meningitis, or osteomyelitis, but more frequently occurs as a localized skin infection. The infection rate closely parallels the colonization rate, so that steps should be taken to decrease colonization. Different approaches to this were looked at in a study at Hermann Hospital during the years 1972–1974. As the umbilical cord is colonized early, preventive measures were considered in an attempt to minimize this colonization. The colonization rates were evaluated with different care protocols. When babies were bathed with 3 percent hexachlorophene, the colonization rate was 22 percent. When the babies were bathed with a 1 percent hexachlorophene solution and a 3 percent solution was applied to the cord, the rate increased to 65 percent. The application of triple dye to the umbilical cord reduced the colonization rate to 16 percent. The infection rate was 0 with the use of triple dye and as high as 15 percent of those colonized during the hexachlorophene use, demonstrating that triple dye is an effective preventive measure for staphylococci.[19] After admission to the nursery, infants have the umbilical cord and skin for 1 cm around the cord painted with triple dye. There have been no reported toxicities to triple dye.

Congenital syphilis cannot be prevented by good nursing care, but the sequelae can be minimized if detected early. Syphilis is acquired *in utero* by transplacental passage of the organism and can cause a variety of problems depending on the trimester in which it is contracted. Almost all infants born to mothers with untreated infection will have the disease at birth. If treated early, many of these infants will escape central nervous system involvement. Because many of the infants will appear normal, the only way to approach the diagnosis is through a screening examination. Each newborn should have a serologic test for syphilis on the cord blood or infant blood.[20]

Harmful Internal Processes

The prevention of hemorrhagic disease of the newborn is an important part of care. This term was first used in 1894 by Townsend, when he described 50 cases, 31 of whom died, with bleeding on the second or third day of life. Hemorrhagic disease of the newborn is caused by a deficiency of vitamin K; and as Vitamin K is required for the liver production of coagulation factors II, VII,

IX, and X, its deficiency causes bleeding. The diagnosis is made by the finding of a prolonged prothrombin time and a partial thromboplastin time. It was noted in the early 1930s that the incidence of hemorrhage could be decreased by starting supplemental feedings at 4 hours of age. In 1939, it was proven that vitamin K could prevent and cure the hypoprothrombinemia in newborns. In 1942, it was demonstrated that cow's milk had 4 times the amount of vitamin K of breast milk, thus explaining the benefit from early feedings in formula-fed babies. Because of irregularity of feedings and absorption, however, the American Academy of Pediatrics recommends a single parenteral dose of vitamin K preparation. This is given as 0.5–1.0 mg of vitamin K soon after birth. The use of vitamin K has eliminated hemorrhagic disease of the newborn.[21]

The next area of protection is recognition of hyperbilirubinemia. The importance of recognizing significant hyperbilirubinemia is that bilirubin, if present at abnormal levels, can result in significant brain damage, or kernicterus. Serum-unconjugated bilirubin concentrations will exceed 2 mg% during the first week of life in approximately 90 percent of newborn infants. Moderately elevated bilirubin levels in normal newborns are so common that the term *physiologic jaundice* has been used. Severe hyperbilirubinemia results when there are factors in addition to the physiologic limitations present in the normal neonate. The diagnosis of physiologic jaundice may be considered in any newborn with mild unconjugated hyperbilirubinemia but is only established by excluding known causes of neonatal jaundice. To learn the approach to hyperbilirubinemia, it is first important to understand the basics of bilirubin metabolism.

Bilirubin is derived from multiple sources, but 80 percent results from the breakdown of the hemoglobin of mature, circulating erythrocytes. This unconjugated bilirubin then is transported in plasma bound to albumin. This bilirubin-albumin bond is important because only that bilirubin *not* bound to albumin is available to cause central nervous system toxicity. As the plasma carries the bilirubin to the liver, it then enters the cells by carrier-mediated diffusion; and two hepatic cytoplasmic proteins, Y and Z proteins, serve as intracellular acceptors of bilirubin. The bilirubin is then conjugated with glucoronic acid for excretion into bile ducts and then into the intestine.[22]

As stated before, a diagnosis of physiologic jaundice can be considered only after ruling out pathologic jaundice. This is accomplished by the appropriate evaluation, which includes a physical examination of the baby for any signs of infection and a minimal evaluation by the laboratory. This includes the measurement of the

hemoglobin, a reticulocyte count, a blood smear, bilirubin measurement (total and direct), blood and Rh typing, and Coombs' testing. This information will help diagnose isoimmunization, frequently the cause of jaundice seen in the first 24 hours of life. As Rh disease has been minimized with the use of RhoGAM, the more common problem now is ABO incompatibility. The laboratory studies may show decreased hemoglobin level and increased reticulocyte count. The smear will show nucleated red blood cells and microspherocytes. The blood types will be incompatible (the mother will be O, the baby A or B), and there is often a positive Coombs' test. At times in ABO disease, the Coombs' test may be negative, but heat elution to remove the antibody from the red blood cell will make the diagnosis. Nonisoimmunization causes of hemolysis should be considered if the picture is compatible with hemolytic disease with a negative work-up. These causes include red blood cell enzyme deficiencies such as pyruvate kinase deficiency and membrane abnormalities such as spherocytosis.

Another important cause of jaundice is enclosed hemorrhage. This may be obvious, as in a cephalhematoma, but it may also be hidden in the head or abdomen, for example. A helpful clue in this diagnosis is a falling hematocrit level if the blood loss is significant. Metabolic causes of jaundice include galactosemia, an inborn error of metabolism, which can be diagnosed by screening the urine for reducing substances. As galactose and glucose are both reducing sugars, the presence of a positive Clinitest tablet (indicating the presence of a reducing sugar) and a negative dipstick (indicating the absence of glucose) would be an indication for further evaluation for galactosemia.

After appropriate evaluation for hyperbilirubinemia is complete, then therapy may be indicated. The American Academy of Pediatrics has established guidelines for exchange transfusion in the event of significant bilirubin-level elevation. FAGA infants are exchanged at an indirect bilirubin level of 20, but this number may be lowered in special circumstances.[15] Conservative measures to try to prevent exchange transfusion include insuring adequate hydration and use of phototherapy. The use of phototherapy was popularized after an observation that bilirubin levels decreased in infants exposed to sunlight.[23] There are many concerns about potential hazards because *in vitro* studies have suggested cellular damage as a result of phototherapy. Though this issue remains unresolved, phototherapy is widely used and accepted. It is important to remember when using phototherapy that the skin color improves while the serum bilirubin level may remain elevated, so that levels must be

monitored even though the baby appears less jaundiced. The immediate complications of phototherapy are related to its inherent property as a heat source. Babies' temperatures should be monitored at least every 4 hours to assure they are not overheated. As the insensible water loss is increased under phototherapy, infants should be weighed at least twice daily to help evaluate fluid status. Urine output and urine specific gravity are also important indicators of hydration. If infants are not receiving adequate fluids, their weights will decrease, their urine outputs will decrease, and their urine specific gravities will increase. These are signs for the nurse to increase fluid intake.

Another harmful influence is the group of disorders known as inborn errors of metabolism, or errors in amino acid metabolism in which tremendous damage occurs in the infants as a result of enzyme deficiencies. These deficiencies result in abnormal concentrations of normal products. The one for which screening is required by law in 44 states is phenylketonuria (PKU), which occurs in 1 of 15,000 births. PKU is an autosomal recessive disorder characterized by mental retardation, with most untreated patients having IQs of less than 30. These children are normal at birth because the phenylalanine does not collect *in utero*. After birth, when feedings are started, the phenylalanine, which cannot be broken down, accumulates and begins to cause pathology by altering biochemical differentiations in the brain. The treatment is to minimize phenylalanine in the diet; and when this is done before 2 months of age, the prognosis for the child is good. It has been calculated that there is an IQ loss of 5 points for every 10 weeks without treatment,[24] so it is thus important to diagnose this disorder early. To understand the optimal way to diagnose PKU, the basics of phenylalanine metabolism must be understood.

Phenylalanine, which is an essential amino acid for protein synthesis in mammalian tissue, is derived from dietary protein and tissue protein. The most important degradation pathway is through hydroxylation to tyrosine. Tyrosine is also an essential amino acid for protein synthesis, as it is a precursor for biogenic amines such as dopamine and norepinephrine (important in the nervous system) and as a substrate for thyroxine and melanin synthesis. The enzyme responsible for this hydroxylation process is phenylalanine hydroxylase.

In the presence of a block in metabolizing phenylalanine, alternate pathways are sought. These include transamination of phenylalanine to phenylpyruvic acid. This pathway is induced by sustained high levels of phenylalanine. Understanding this is the basis for diagnosis. The key is to determine elevated phenylalanine in the blood. This

can be done several ways, but timing is important. As dietary intake is the source of phenylalanine, PKU testing must be done only after the establishment of feedings containing phenylalanine. It is routine to test after 24 hours of milk feedings, but this is not optimal because there is variation in the rise of phenylalanine in PKU babies and the variation may be a sex-determined factor (baby girls with PKU are more likely to be missed with early newborn screening than baby boys). Also, early discharge from the hospital is now more common. With current newborn screening, between 5 and 10 percent of those with PKU are not detected. The optimal screening time would be at approximately 7–14 days of age, so a system needs to be established for adequate follow-up to assure that all babies have the PKU testing done. The nurse can be very helpful in educating the parents on the importance of this follow-up. Some have proposed that use of phenostix (urine dipsticks) provides adequate screening, but this is probably not so. The urine test is based not on phenylalanine in the urine but on the presence of the products of alternate pathways for phenylalanine metabolism. As I have explained, these alternate pathways are induced by sustained levels of phenylalanine and therefore the results of such tests will not be positive until late in the course of the disease.

The most common blood test is an inhibition assay that is based on the inhibition of growth of the organism *Bacillus subtilis* by a phenylalanine analogue. If the blood sample contains more than a certain concentration of phenylalanine, inhibition is overcome and the organism grows. Correct collection of the blood is essential in view of this technique. The filter paper usually has circles on it, and these must be filled completely with blood or the volume of blood may be too small even in the face of an elevated phenylalanine level. False positive results occur frequently in PKU testing, and 85 percent of all newborns initially showing positive results will be normal. This is important to recognize because placing an infant who does not have PKU on the low-phenylalanine diet can be hazardous. Tests should be repeated in all instances of positive results; and if they remain positive, a referral should be made to a tertiary care center for further evaluation and therapy.[24]

PROMOTION OF GOOD HEALTH IN TRANSITION TO EXTRAUTERINE LIFE

One of the most important aspects of FAGA newborn care is promoting maternal-infant bonding. This bond is critical for the health of newborns because they are totally dependent on their

mothers for nurture, both physical and emotional. Interruptions in this process by separation of mother and infant can have long-lasting effects and may result in failure to thrive or battering. Although separation does not occur with FAGA infants, other problems can interfere with bonding, and some of these are routine hospital practices. The nurses must help promote early contact between mother and baby. Klaus and Kennell have designated the period immediately after birth as the "maternal sensitive period."[25] This period is that time in which the parents become attached to their infant.

Attachment can be promoted in the delivery room by allowing the mother to hold the infant after initial stabilization and by giving her the opportunity for eye-to-eye contact with the baby. She can also be allowed to nurse the baby at this time. Joint recovery rooms in which the mother and baby go through the transitional period together can be helpful as well. Rooming in is also an important part of promoting attachment, as it allows the mother and baby prolonged hours alone, a situation that might not be possible after discharge from the hospital and return to the family. Guidelines for rooming in can be found in the American Academy of Pediatrics' *Standards and Recommendations for Hospital Care of Newborn Infants.*[15]

SUMMARY

The nurse is a critical member of the health care team. FAGA infants are by definition at low risk for problems and require minimal attention from a physician. The nurse who spends a great deal of time with them and their mothers is in a position to prevent many problems FAGA infants could develop and also to recognize problems when they do occur.

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