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Neonatal Nutritional Care

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Nutritional Care in the Neonatal Intensive Care Unit:

A *premature birth* is defined as a birth occurring at or before 37 weeks of gestation (1), but it encompasses so much more than just gestational age. A premature infant is physiologically different from a full-term infant. The physiological differences bring about a host of medical problems and disorders, which must be handled differently from those of full-term infants. However, little research exists to directly address the question of how to treat premature infants, especially when determining their nutritional needs. It would be unethical and difficult to conduct research with such a high-risk group, but these infants do need to be examined carefully to ensure the best possible care is provided to them. The purpose of this project is to describe the nutrition-related problems associated with prematurity, and then to use a series of case studies to illustrate these nutrition-related problems and their possible solutions.

First, statistics relating to preterm birth will be given to demonstrate the significance of prematurity as a public health problem. In addition, the causes of premature birth and goals for the prevention of premature birth will be outlined. In order to understand preterm birth, a brief review of normal physiology and development has been included. In addition, an overview of premature infant care will be given. After that, background information about nutritional care and common medical disorders will be explained in detail. This will set the stage for the explanation of how premature infants are cared for in a hospital setting. The nutrition-related problems for five premature infants will be described, followed by an explanation of each infant's treatment. The case studies will be used to set

general guidelines for how the nutritional demands of a premature infant should be met.

Statistics

Prematurity is a severe public health problem. When the statistics are examined, the breadth of the problem becomes more apparent. More than 470,000 babies are born preterm each year in the United States. The incidence of prematurity has increased by 27% since 1981 and it accounts for 12% of all live births (2, 3).

Prematurity is the leading cause of perinatal mortality (death during the time period of 28 weeks gestation through six days of life), neonatal mortality (death through the time period of zero through 27 days of life), and the second leading cause of infant mortality in the United States. Preterm birth leads to neurodevelopmental handicaps, chronic respiratory problems, intraventricular hemorrhage, periventricular leukomalacia, infection, retrolental fibroplasia, necrotizing enterocolitis, and neurosensory deficits (2, 3).

The financial costs of prematurity are staggering. The average lifetime medical expenses for a preterm baby are \$500,000. In 2001, 29 billion dollars were spent on hospital charges for all infants. Over half of this figure can be attributed to prematurity. The average cost of a hospital stay for a preterm baby is \$75,000, compared to \$1,300 for an uncomplicated newborn stay. Figures 1 and 2 demonstrate that, although premature births account for only a small percentage of infant hospital stays, they account for a larger percentage of hospital charges incurred annually (4).

<u>Risk Factors for Preterm Labor</u>

Preterm birth is categorized into three types: *spontaneous preterm labor*, which accounts for 50% of all preterm births; spontaneous premature rupture of the membranes, which accounts for 25% of all preterm births; and medical *intervention*, which accounts for 25% of all preterm births (5). The risk factors for each of these categories are quite similar. The most prominent risk factors for preterm birth are multifetal pregnancy, a history of preterm labor or low birthweight, repeated mid-trimester bleeding, and uteral, placental, or cervical abnormalities. Maternal age also plays a determining role in the incidence of preterm birth. Maternal age less than 17 years or over 35 years increases the risk for preterm labor. Maternal obesity or low pre-pregnancy weight can also increase the risk for preterm labor (5). Other risk factors include black race, low socioeconomic status, infections, anemia, bleeding, major stress, alcohol abuse, illicit drug use, and folic acid deficiency. Folic acid deficiency can lead to neural tube defects, and other birth defects such as cleft lip/palate, cardiac defects, limb reduction, and urinary tract defects. Other problems associated with folic acid deficiency are atherosclerotic vascular disease, colorectal and cervical cancer, acute lymphocytic leukemia, and Alzheimer's disease (5).

Behaviors involving tobacco and alcohol use during pregnancy usually result in adverse outcomes. Smoking during pregnancy contributes to 20% of all low birth weight incidents, 8% of preterm births, and 5% of all perinatal deaths. Smoking also increases the risk of stillbirth and sudden infant death syndrome (SIDS). In a case-control study conducted with 741 mothers, 174 of whom were smokers and 567 of whom never smoked, maternal smoking was associated with a mean birth weight reduction of 377 grams (6). Alcohol during pregnancy can lead to miscarriage, premature birth, low birth weight, fetal alcohol syndrome (FAS), alcohol-related birth defects (ARBD), alcohol-related neurodevelopmental disorders (ARND), and birth complications (5).

Goals for Preterm Labor Prevention

The ultimate goal in reducing the incidence of preterm labor is primary prevention, especially during the preconceptional period and early in pregnancy. Already existing prevention programs have focused on risk assessment or prediction of preterm labor, but risk assessment only identifies half of all preterm births. Primary prevention involves first identifying and managing risks, and then trying to reduce risks and improve reproductive health. Secondary prevention involves the prevention of preterm labor, and tertiary prevention involves minimizing the complications of prematurity (5). Interventions that actually work are quite comprehensive. First of all, there must be early prenatal care accessible to all pregnant women. All pregnant women ought to be educated about preterm labor signs and symptoms. In addition, all urinary tract infections (UTI) and sexually transmitted infections (STI) should be treated immediately. Interventions should be initiated for smokers, alcohol-users, and illicit drug users. Folic acid deficiency should be eliminated, and mothers should be screened for domestic violence. Stress levels should be reduced throughout pregnancy. Optimal weight should be stressed, and exercise and activity should be a part of daily life (5). It has been documented that poor nutritional status is a cause of low birth weight infants. Over 50% of low birth weight births can be attributed to the mother's nutrition status (7).

The March of Dimes has launched a campaign with the goals of increasing public awareness of the problems caused by prematurity and decreasing the rate of preterm birth in the United States by at least 15%. In meeting these goals, educating pregnant women and making healthcare available to all are major priorities. Eliminating preventable risk factors, such as smoking and alcohol or drug use, are also top priorities for the March of Dimes (5).

Normal Physiology and Development

The main problem of preterm birth is the physiological difference between preterm and full-term infants. Comprehension of normal physiology and development is necessary to understand prematurity. The growth and development of a fetus occurs based on the genetic code, which controls the rate at which cells multiply, differentiate, and attain their long-term functional status. These changes occur at set time periods, called *critical periods*. The most serious critical periods occur during the first two months after conception, which is when the majority of organ systems begin to take form. Once a critical period is complete, the results cannot be reversed. Therefore, if an error occurs during a critical period, it cannot be corrected. This sets the importance of proper nutrition and other factors during pregnancy. Critical periods are defined by hyperplasia, which is rapid cell multiplication (8).

The first organ to develop is the brain. It is the organ that gets the first consideration for energy and nutrient needs. The energy requirements of the

central nervous system will be met first during times of low energy availability. An energy deficit or even an excess of nutrients can result in permanent damage, such as defects in organ structure and function. The organ or tissue experiencing a critical period of growth will be affected the most at the time of insult. The neural tube develops into the brain and spinal cord during the third and fourth week after conception. If there is a folate deficiency at this time, there are irreversible defects in brain and spinal cord formation (8).

After hyperplasia occurs, hypertrophy usually occurs. Cell multiplication will slow down, and the size of the cells will increase. In addition, the specialized function of cells will appear. Cells become more stable in this phase, with the number and size remaining constant (8).

The First Trimester

During the first trimester, formation of the three embryonic tissue layers occurs. The three embryonic tissue layers are the endoderm, the mesoderm, and the ectoderm. The endoderm is the innermost layer, which will eventually differentiate into the respiratory tract, digestive tract, bladder, and urethra. The mesoderm is the middle layer which, will differentiate into connective tissue, bone, muscle, circulatory system, kidneys, sexual organs, and the lymphatic system. The ectoderm, the outermost layer, will differentiate into epidermal and neural tissue. By the third week of gestation, organs begin to differentiate from the original cells, and the major organ systems develop during the second month. By the eighth week, the gene pathways that determine the sex of the fetus are turned on, allowing for the development of sex organs (9).

The Second Trimester

The second trimester characterizes a time of growth for the fetus. The main outcome is an increase in size and the development of bones. The fetus will begin to move during this trimester (9).

The Third Trimester

The third trimester also involves an increase in size of the fetus. The circulatory and respiratory systems develop in order to prepare the fetus for breathing outside of the uterus. At this time, antibodies from the mother will be passed to the fetus, providing the fetus with temporary immunity (9). The third trimester is most affected by preterm birth. Since preterm birth means a shortened third trimester, the fetus does not have enough time to prepare for life outside of the uterus.

Newborn Assessment

The first assessment of newborn health is determined by the APGAR score (1). This is a rating score that is assigned at one minute and five minutes after birth. APGAR is acronym for activity (muscle tone), pulse, grimace (reflex irritability), appearance (skin color), and respiration. The lowest possible score is zero, while the highest is ten. Each category for assessment is given a score based on its presence and intensity. For example, activity is given zero points for the absence of muscle tone, one point for the arms and legs being flexed, and two points for active motion. Infants with low APGAR scores generally have undergone stress during labor and delivery. Infants generally do not have between four and seven may need some resuscitation. Infants with a score of three or below will need immediate care (1).

Guidelines for Premature Infant Care

Premature infants spend the first part of their lives in the neonatal intensive care unit (NICU) under the close watch of neonatologists and nurses. The American Academy of Pediatrics (AAP) has set guidelines for the care of extremely premature babies, to try to address all medical, social, and ethical factors that impede the infant's care (10). Frequent evaluations must be performed by the physician to make proper decisions for the infant's care involving the parents. In addition, the physician must remain up-to-date on all treatment options for the infant, in order to provide optimal care. The physician must share all relevant information with the parents of the infant to avoid any confusion and to help them in making decisions about their child's care (10). The AAP has also set guidelines involving the discharge of the infant from the NICU. Premature Infant Discharge

Infant discharge from the hospital must be determined based on a variety of factors (11). It is difficult to decide when to discharge a premature infant because of the conflicting issues of the high cost to keep an infant in the NICU and the intense medical care needed. The benefits of early discharge include less separation time between the infant and the family and a decreased risk of morbidity associated with hospital procedures (11). However, it is very difficult to assess physiological stability, which is the main determinant for readiness for discharge. Investigations of medical records have shown that preterm infants who spent any time at all in the neonatal intensive care unit have a higher hospital readmission rate and death in the first year of life when compared to full-term infants. The preterm infant is classified as one of four categories of high risk situations that require extra attention when determining hospital discharge. The other three categories are: [1] the infant who requires technological support, [2] the infant primarily at risk because of family issues, and [3] the infant whose irreversible condition will result in an early death (11).

The previous requirement for determining discharge from the hospital was the achievement of a preset weight of five to five and a half pounds (11). However, research has shown that other factors are more important than the achievement of a specific weight. A regular pattern of weight gain, the ability of the infant to thermo regulate without mechanical help, the ability of the infant to suckle feed, assessment of the home, parental preparation, and frequent visits and follow-ups after the infant has been discharged are other factors that are just as important, if not more important, than a set weight (11). Current recommendations involve educating the parents on care and how to prepare their home for a high-risk infant. In addition, the infant must have received all necessary immunizations and have undergone all appropriate health and nutrition screenings (11).

Characteristics of Low Birth Weight Infants

The gestational age of an infant is the age of an infant at birth, based on the mother's last menstrual period (1). The gestational age is an important tool for assessing physiological function and nutrition status. It can be determined by a series of clinical assessment criteria, including neurological factors and external features that indicate the physical maturity of the infant. Gestational age provides a basis for another way of classifying infants. A small-for-gestational-age (SGA) infant weighs less than the 10th percentile of the standard weight for that gestational age. An appropriate-for-gestational-age (AGA) infant has a birth weight between the 10th and 90th percentiles on the intrauterine growth chart. A large-for-gestational-age (LGA) infant weighs more than the 90th percentile on the intrauterine growth chart (1).

The preterm infant has a diminished nutritional status at birth because fetal stores of nutrients are built up during the last three months of pregnancy. Preterm infants lose a portion of this last three months, depending on how early they are born (12). This means that nutritional support needs to begin as soon as possible. The preterm infant weighing 1,000 grams has only one percent of body weight as fat. The term infant weighing 3,500 grams has 16% of body weight as fat. Preterm infants have an increased basal metabolic rate, due to their rapid rates of growth and development, and they will quickly deplete their limited reserves. However, underdeveloped organ systems and medical problems hinder the feeding process. Moreover, malnutrition exacerbates the infant's medical problems. Intravenous feeding becomes very important in these situations (12). Nutritional Care

While the optimal nutritional care plan has not been determined, the general consensus is that the use of human breast milk is the most favorable option (13). Human milk is tolerated earlier than other forms of nutrition for very

low birth weight infants. It also enhances the development of the gastrointestinal tract, while accelerating gastric emptying and reducing gastric residuals. The incidence of necrotizing enterocolitis is reduced when human breast milk is administered. This is an important fact, since necrotizing enterocolitis is a significant problem among preterm babies requiring immediate care (13). Necrotizing enterocolitis is a condition characterized by the death of intestinal tissue and it can lead to perforation, bowel resections, and long-term growth and feeding problems that are related to a shortened bowel. The antiinfective characteristics of human milk are another reason to choose human milk over formula. Infection and sepsis/meningitis rates have been found to be lower among infants who have been fed human milk, as opposed to those fed formula. Human milk contains immunoglobulin A, lactoferrin, lysozyme, oligosaccharides, growth factors, and other cellular components. The milk also contains antibodies that can protect the infant (13).

Infants that have been breast fed also experience fewer incidents of gastroenteritis, respiratory infections, otitis media, urinary tract infections, and sepsis. Immune factors are the most likely reason for fewer incidents of these conditions (13).

Even though the benefits of breast milk are compelling, it alone does not meet the nutritional requirements of extremely low birth weight infants. After discharge, infants fed nutrient-enriched formulas experienced greater growth gains than those that were breast fed (14). Human milk fortifiers can be added to human milk to increase the caloric value of the milk and add nutrients to the milk. The human milk fortifier results in greater weight gains than those not receiving human milk fortifier. Furthermore, with the human milk fortifier, the weight gain occurs as the accumulation of lean body mass. It is also suggested that human milk fortifier can increase bone mineral content (13). The most popular human milk fortifier used in NICUs is made by Enfamil, which is a Mead Johnson company.

Demand feeding is a method of nourishing an infant based on hunger cues (15). Demand-fed infants have been shown to exhibit signs of clinical improvements as compared to infants who were nourished by other feeding patterns (15). In addition, demand-fed infants had shorter hospital stays, improved weight gain, and better behavioral responses relating to feeding (15). While the research supporting demand feeding is limited, it suggests a useful way of nourishing high-risk infants.

The nutritional care plan determined for each infant is based on the infant's overall health status. One key indicator of health status of the infant is weight. The average gestation of a full-term infant is 40 weeks, and the range is 37 to 42 weeks (1). The average weight for a full-term infant is 2,500 to 3,800 grams (5.5 to 8.5 pounds). A preterm infant is defined as an infant born at or before 37 weeks of gestation. A low birth weight infant weighs less than 2,500 grams (5.5 pounds) at birth. A very low birth weight infant weighs less than 1,500 grams (3 pounds 5 ounces) at birth. An extremely low birth weight infant weighs less than 1,000 grams (2 pounds 3 ounces) at birth (1). Once a preterm infant's weight is measured, the infant is classified into one of these categories.

Certain risks are associated with low birth weight, and the risk levels increase as the weight decreases. Very low birth weight infants experience more growth problems and undernutrition, despite efforts to provide adequate nutrition (13). This puts the infants at a greater risk for cognitive and motor abnormalities later in life. In addition, these infants generally have a higher hospital readmission rate (13). In order to adequately nourish these infants, special considerations should be given to the nutritional care plan.

Parenteral nutrition is the intravenous provision of nutrients directly into the bloodstream (16). Parenteral nutrition can be administered via a central route or peripheral route. Central access involves the insertion of a catheter tip into a large vein with a high amount of blood flow, such as the superior vena cava. This type of parenteral nutrition is called total parenteral nutrition (TPN). Peripheral access involves the insertion of the catheter tip into a smaller vein usually in the arm. However, TPN is the preferred method of parenteral nutrition since it seems to be most effective in improving nutritional status (16).

Parenteral nutrition solutions are made containing amino acids, carbohydrates, and lipids. Standard solutions for protein contain both essential and nonessential crystallized amino acids. Specialized solutions are made for patients with specific conditions requiring a certain proportion of amino acids. Amino acid concentrations range from three to 15%. There are approximately 4 kilocalories per gram of protein in an amino acid solution (16).

Carbohydrate is administered as dextrose monohydrate in concentrations ranging from five to 70%; this provides 3.4 kilocalories per gram. Carbohydrate

in a parenteral nutrition solution spares protein as an energy source. Carbohydrate administration must be carefully monitored to prevent hyperglycemia, hepatic abnormalities, and increased ventilatory drive. The osmolarity of a solution should be calculated to avoid venous intolerance (16).

Lipid emulsions are used to provide calories in the form of lipid. They are composed of aqueous suspensions of soybean or safflower oil, with egg yolk phospholipid as the emulsifier. They are provided in 10% and 20% concentrations. The 10% solutions supply 1.1 kilocalories per gram and the 20% solutions supply 2.0 kilocalories per gram. The lipid emulsions contain glycerol, which is water soluble. Glycerol supplies osmolarity to the solution and it can be oxidized, providing 4.3 kilocalories per gram (16).

Electrolytes, vitamins, and minerals must be given in parenteral solutions to meet daily requirements. However, the amounts of vitamins and minerals given must be lower than the Daily Recommended Intakes (DRIs) because they are supplied directly into the blood stream, meaning they avoid all digestive and absorptive processes. Parenteral solutions are the major source of daily fluid and electrolyte intake. Therefore, they must be adequately prepared to keep the patient in fluid and electrolyte balance (16).

The ultimate goal for nutrition is to use the gastrointestinal tract without causing any problems. Therefore, there is a period of transitional feeding after parenteral feeding to start enteral, or oral, feeding (16). With preterm infants, enteral feeding is the next step after parenteral feeding. During transitional feeding, patient tolerance must be closely monitored. The major difficulty is providing enough calories to meet energy requirements. That means there should be calories supplied by both parenteral and enteral nutrition. The first step in the transition from parenteral to enteral feeding is to administer a small amount of enteral feeding at a low rate to establish tolerance (16). Once there have been no signs of intolerance after several hours, the enteral rate should be increased every few hours while reducing the parenteral rate in accord. When 75% of the energy needs can be met by enteral nutrition, parenteral nutrition can be discontinued altogether. However, with preterm infants, extra care must be taken. There is usually a multitude of disorders affecting the infant and this will impede the enteral feeding process. It is possible that during the transition period, a new problem can arise, causing feeding intolerance, and the infant will have to revert back to TPN (16).

Enteral nutrition is essentially tube feeding. It is preferred to parenteral nutrition because the gastrointestinal system is accepted as the metabolically and physiologically better way of obtaining nutrition. The route of access for enteral nutrition is selected based upon many factors, including predicted length of enteral feeding time, level of risk for aspiration, presence of normal bowel sounds, the necessity of surgery, and properties of the enteral formula. Several routes are used; an example is the nasogastric route, which is used for short-term nutrition. In this case, the tube is passed through the nose into the stomach. Another example is the orogastric route, which involves tube placement into the mouth then down into the stomach (16).

Common Medical Disorders

Preterm infants have not had adequate to complete proper intrauterine development. Therefore, they are physiologically different from full-term infants. There are many systemic problems that occur in the preterm infant. The most common respiratory problems are respiratory distress syndrome and chronic lung disease, also known as bronchopulmonary dysplasia. Patent ductus arteriosus is a major problem for the cardiovascular system. Fluid and electrolyte imbalances pose severe detriments for the renal system. Intraventricular hemorrhage and periventricular leukomalacia are the most common neurologic problems. Hypoglycemia, hyperglycemia, hypocalcemia, and metabolic acidosis often occur in preterm infants, creating metabolic problems (1, 9).

Congenital Heart Disease:

Congestive Heart Failure

Congestive heart failure (CHF) is one type of congenital heart disease that plagues a premature baby. Fluid builds up in the lungs, which leads to respiratory distress and edema. Therefore, the cardiac and respiratory workloads are both increased, along with an increased respiratory rate (17). All of this translates into increased metabolic needs for the neonate. A baby born with congestive heart failure needs approximately 30% more energy than a baby born without a heart condition. With CHF, there is an elevated risk of infection, as well as a reduction in the amount of blood flow to the peripheral tissues. Other complications include increased protein loss and decreased nutrient absorption (17). Diuretics can be used to help eliminate the extra fluid in the heart, and Digoxin can be used to amplify the strength of heart contractions. On top of that, the baby may need fluid and sodium restrictions. CHF causes increased emesis and tachynea. The infant will tire quickly and be unable to eat much in one sitting. This means that the infant must be given small, frequent feedings in the form of concentrated, low sodium formulas. The infant may require oxygen with feedings. If the problem persists, surgery may be needed (17).

Patent Ductus Arteriosus

All babies have a ductus arteriosus. It is an open passage for blood flow between the pulmonary artery and aorta, which usually closes within a few hours after birth. Since the fetus uses the placenta to obtain oxygen, the lungs can be bypassed. This occurs via the ductus arteriosus (17). Premature babies often have no yet reached the stage of development where the ductus arteriosus closes after birth. This is a condition known as patent ductus arteriosus (PDA). The total body oxygenation decreases when the ductus arteriosus stays open (17). With a larger ductus, the infant tires easily and grows slowly. Pneumonia and fast breathing are common with PDA. Medications such as Indomethacin or surgery are used to close the ductus. Fluid intake must be restricted, and enteral nutrition must be discontinued because of decreased gut perfusion. Once the ductus is closed, enteral nutrition can be used once again (17).

Central Nervous System Disorders:

Intraventricular Hemorrhage

Intraventricular hemorrhage (IVH) occurs as a result of the especially small and fragile blood vessels in a preterm infant's brain. These blood vessels can rupture near the ventricular lining. When this occurs, it is usually during the first three days of life. Thirty to 40% of infants weighing less than 1500 grams experience IVH. It can lead to severe neurodevelopmental handicaps in extremely low birth weight infants. The immediate complication involved with IVH is hydrocephalus, which is an abnormal accumulation of fluid within the ventricles of the brain. In order to treat this problem, a shunt is inserted to channel the fluid away from the brain or spinal cord to another part of the body (17).

Myelomeningocele

Myelomeningocele is better known as spina bifida. It is a neural tube defect where the neural tube does not close, causing a sac to develop. This usually occurs at approximately 26-30 days of gestation. The result can be hydrocephalus, epilepsy, and brain stem malformation involving reflux and poor reflexes (gag, suck, and swallow). Obesity often occurs secondary to decreased metabolic rate. There are often swallowing and chewing problems, leading to feeding delays. Constipation can also be a problem. Currently, no specific treatment exists for spina bifida. Prevention, involving the use of folic acid supplements, is the main objective for healthcare professionals (17). Gastrointestinal Disorders:

Dehydration and Diarrhea

An immature gut cannot properly absorb water and electrolytes. When the concentration of electrolytes in extracellular fluid is high, more water will rush to

the extracellular fluid to dilute it. Diarrhea is the result, since there is an excess of water in the feces. This leaves the body dehydrated (17).

Esophageal Atresia/Tracheoesophageal Fistula

This developmental problem occurs in the upper gastrointestinal tract. Normally, there is a division into two pathways: one that leads to the trachea for the respiratory system and one that leads to the esophagus for the digestive system. In this condition, there is no direct access to the stomach until surgery is performed. TPN must be administered using a central line. If possible, enteral nutrition can be administered using percutaneous endoscopic gastrostomy (PEG), which is the surgical placement of the feeding tube into the stomach (17). *Omphalocele and Gastroscisis*

Omphalocele and gastroscisis are abdominal wall defects where the internal organs are externalized through an opening in the abdominal wall. Omphalocele is a herniation of the abdominal contents through the umbilical cord membrane. The contents are covered by the peritoneal membrane (17).

Gastroscisis occurs when the abdominal contents are externalized to the right of the umbilical cord, meaning there is no membrane enclosing them. Saline solution must be used to keep the abdominal contents moist and they must be covered with plastic. TPN must be administered with an increased amount of fluids since there is substantial fluid loss through the exposed intestines. Gastric suctions will be required after surgery to insert the organs, and TPN must continue until gastric suctioning is complete. Enteral nutrition (EN) should be introduced slowly when normal bowel sounds begin and gastric output is minimal. The appropriate EN formulas are Pedialyte, breast milk, or half-strength formula (17).

Meconium Ileus

Meconium ileus is an obstruction of the terminal ileum caused by a sticky, thick plug of meconium that often requires surgery for removal. It is associated with cystic fibrosis. The result is poor growth due to malabsorption of nutrients, most notably fat, protein, and fat-soluble vitamins. Feedings of predigested formulas, such as Pregestimil, with a high medium chain triglyceride content, should be administered. Fat soluble vitamins should be supplemented with 4,000 units of lipase for each feeding (17).

Necrotizing Enterocolitis

Necrotizing enterocolitis (NEC) is a condition where the cells lining the wall of the intestine slough off and die. The exact cause of NEC is not understood. It is however, associated with multiple factors, including prematurity, infection, hypertonic feedings, hypovolemia, birth asphyxia, hypothermia, ischemia secondary to patent ductus arteriosus, and possibly early enteral feedings or milk protein allergy. The affected areas are usually the jejunum, the ileum, and the colon. In some cases, intestinal perforation occurs, requiring surgery and intestinal resection. One symptom of NEC is feeding intolerance, which is characterized by abdominal distention, large residuals, vomiting, diarrhea, or blood in stools. Respiratory complications, lethargy, apnea, elevated body temperature, sepsis, shock, peritonitis, and increased white blood cell count are all other symptoms of NEC. The infant is NPO, meaning nothing

can be consumed using the gastrointestinal tract, until NEC is ruled out. Parenteral protein needs increase by 10 to 30% (17).

Short Bowel Syndrome

A normal term infant's intestine is 250 to 270 centimeters long. Surgical resection of bowels may be necessary because of congenital problems or necrotizing enterocolitis. The surgeon will try to preserve the ileum and ileocecal valve as much as possible. However, there is a still a significant loss of intestinal surface area, causing the villi to atrophy, and loss of brush border enzymes. The result is inadequate digestion and absorption. When less than 50 centimeters remain, there are extreme limitations of intestinal function (17). The problems that arise are malabsorption, intestinal dysfunction, fluid and electrolyte imbalance, reduced absorptive surface of the intestine, and altered motility through the intestine. For the first 14 days following surgery, the infant must be NPO with TPN. Small EN feedings should be initiated as soon as possible for villous hyperplasia. Sterile water, D5W, or Pedialyte should be given first, followed by diluted casein hydrolysate, fat modified formulas, such as Progestimil, elemental formulas such as Pediatric Vivonex, or human breast milk. Glucose polymers can be added to formulas to increase the caloric density. Vitamin and mineral supplementation should also be included (17). Liver Disorders:

Liver diseases cause anorexia with increased caloric requirements. EN can be administered through an orogastric tube. Glucose polymers can be used for supplementation if the infant is not gaining weight. If ascites (accumulation of fluid in the peritoneal cavity) is present, PO fluid should be restricted and sodium should be restricted to one to two milliequivalents per kilogram per day.

Diuretics such as furosemide should be used (17). Bile deficiency will cause poor fat digestion and absorption. Medium chain triglycerides should be given, since they do not require bile salts for digestion, as they are directly absorbed into the bloodstream. However, certain long chain triglycerides are essential, so there are two formulas that can be used. Progestimil from Mead Johnson contains 28% of calories as medium chain triglycerides and 7% as long chain triglycerides. The other formula is Portagen from Ross. It contains 3.4% of calories as long chain triglycerides and 34% as medium chain triglycerides, but it can lead to essential fatty acid deficiency. Breast milk can also be used when it is supplemented with medium chain triglycerides. Vitamins A, D, E, and K should also be supplemented (17).

Hyperbilirubinemia

Hyperbilirubinemia is a condition characterized by a yellowish tint to the skin. It is caused by an excess of bilirubin, a byproduct of the breakdown of hemoglobin, in the blood. The fetus has more hemoglobin of a type that has a higher affinity for oxygen than normal adult hemoglobin due the fetus' increased need for oxygen (1). When the infant is born, fetal hemoglobin is no longer necessary and the excess hemoglobin is broken down, and bilirubin is released. Bilirubin is a cell toxin that is normally removed from the blood by the liver (1). However, a premature infant's liver cannot keep up with the rapid rate of fetal hemoglobin breakdown, causing bilirubin to build up in the blood. The result is a yellowish tint to the infant's skin. To treat this problem, phototherapy is used. Phototherapy involves the use of fluorescent lights. The light is absorbed by the skin and converts bilirubin into a water-soluble form that can be removed from the body without going through the liver (1).

Pulmonary Disorders:

Respiratory Distress Syndrome

The fetus begins to produce surfactant around the 24th week of gestation but it is not efficient until the 35th week of gestation. Some premature babies do not produce enough surfactant, so that they have immature lungs with an inadequate number of alveoli. The infant tires quickly due to rapid and strained breathing. Respiration is plagued by irregular breathing and periods of apnea. Energy needs are elevated due to increased respiratory muscle work. Nutritional requirements must be calculated based on the rate of carbon dioxide production from glucose. Essentially, the amount of lipid in the diet must be decreased. Amino acids should be increased to stimulate respiration and prevent apnea (17). *Apnea*

Apnea is condition characterized by the absence of breathing. It occurs when the brain does not signal the lungs to breathe. It is associated with cyanosis and bradycardia. If apnea does not resolve before the infant is discharged from the hospital, the infant will be sent home on methylxanthines (theophylline) or caffeine, with a home cardiorespiratory monitor (17).

Chronic Lung Disease

This disease develops in an infant who has been on a ventilator for a prolonged period of time. On the ventilator, the infant receives a higher than normal oxygen and pressure level. This can do damage to existing lung tissue and increase vascular resistance, making it more difficult for the infant to breathe. This incurs a major risk for severe neurodevelopmental handicaps in extreme low birth weight infants (17).

Ventilators

As a result of respiratory problems, many preterm infants must use ventilators to breathe. The ultimate goal of a ventilator is to uphold sufficient gas exchange while reducing the risk of iatrogenic injury. Each infant must be individually considered before being placed on a ventilator. The type of ventilator must be chosen carefully, and monitoring is very important. In addition, the supplemental therapies being administered to treat other complications must be taken into account when choosing a ventilator (18). Renal Failure:

Perinatal hypoxia leads to renal failure. Decreased amounts of protein must be administered with decreased amounts of fluid to reduce the strain placed on the kidneys. Urinary output decreases with renal failure. There is usually poor weight gain and inadequate intake (17).

Case Studies:

In order to study the actual treatment of premature infants in a hospital setting, the early stages of life were observed for five infants in the NICU at

Westchester Medical Center in Valhalla, New York. Basic data were collected, including biochemical laboratory values and observations of the nutritional and medical treatment provided to the infants. The five infants were observed, keeping all identifiers anonymous. They were identified as Baby A, Baby B, Baby C, Baby D, and Baby F. Initially, another infant called Baby E was part of the observation, but data collection complications required Baby E to be removed from observation.

The normal values of the biochemical data obtained are shown in Table 1. The normal values are given for each of these parameters to assess the physiological function of various organs. Medical disorders are identified based upon the maintenance of laboratory values within normal limits. Blood glucose levels were recorded as an indicator of metabolic function. Blood urea nitrogen is a waste product of muscle breakdown. Elevated levels indicate that more protein is being hydrolyzed than is considered normal, while lower levels indicate that the patient is losing muscle due to an insufficient protein intake (17). Creatinine is another product of muscle breakdown and it is normally present in the blood. When serum creatinine is high, the patient is losing weight and muscle, or the kidneys are not properly working. When serum creatinine is low, the patient has little muscle protein. Sodium in the blood affects hydration and blood pressure. When sodium levels are high, blood pressure will increase because there will be a fluid overload in the blood. This leads to edema and congestive heart failure (17). The excess fluid in the blood will dilute the blood, leading to a multitude of problems. Potassium was recorded since it impacts muscle action, most notably

the heart. When potassium levels are too high, the heart can stop beating. When potassium levels are too low, there is muscle weakness or arterial fibrillation (17). Serum albumin controls the osmotic pressure of the blood. Bilirubin levels in the blood increase as a result of medications, gallstones, biliary duct diseases, intravascular homolysis, or hepatic immaturity. Low levels indicate anemia. White blood cells are a measure of infection, neoplasia, and stress when the count is high. When the count is low, it indicates protein energy malnutrition, autoimmune disease, or an overwhelming infection. Hemoglobin and hematocrit values were recorded as an indicator of nutritional deficiencies, hemorrhage, marrow failure, or renal disease (17).

When examining the case studies, the nutrition-related problems can not always be separated from other medical problems. The treatments often involve nutritional strategies, as well as other medical procedures. The reasons for each nutritional intervention and how the intervention was implemented were examined. The results of each intervention shed light onto the area of nutritional care for premature infants and can be used to make decisions regarding infant care in the future.

Baby A:

Gestational Age: 24²/₇ weeks

Length at Birth: 34 cm

Apgars: One minute: 4, Five minutes: 7

Baby A was a male born at $24^2/_7$ weeks. He weighed 540 grams at birth (1.19 pounds) and was 26.5 centimeters long (10.43 inches). His major problems

at birth were respiratory distress, dysmorphic features, and the possibility of sepsis. He also needed phototherapy for hyperbilirubinemia. These are all common problems that very premature babies face.

Another problem that Baby A faced commonly affects all premature babies. They tend to lose weight within the first few days of life because they lose water. The weight loss usually does not last more than a few days, but it is critical to maintain as much weight as possible (18). Baby A followed the typical pattern of weight loss and was back on track by the sixth day of life.

All of these problems put together create a need for accurate nutritional care. Every other aspect of the baby's care must be taken into account when creating a nutritional care plan. Respiratory distress increases energy needs (19). The baby has to work harder to take in air and get oxygen into the lungs. If the infant is septic, there is also an increase in the need for energy. The immune system is working hard to kill foreign invaders, but it is not at the proper stage of development to be fighting invaders. Sepsis occurs easily in preemies for this reason, and the feeding line for TPN must be carefully monitored and kept sterile.

Baby A started out NPO, which is typical of premature babies. When NPO, the babies receive total parenteral nutrition (TPN). By his fourth day of life, Baby A was able to start enteral feeds of sterile water at a very slow rate, along with the already-existing TPN. The next day, Baby A was administered expressed human milk enterally at a slow rate to test his tolerance. He continued at this rate for a few days, and then advanced as tolerated. By his twelfth day of life, Baby A was no longer receiving any parenteral nutrition. To increase calories, Baby A was given human milk fortifier, which is added to human milk. This plan of care worked for Baby A until data collection stopped. Table 2 details all of the data collected for Baby A. It includes biochemical parameters as well as physical parameters. The most notable biochemical parameters in Table 2 were the high sodium values, indicating electrolyte imbalance, and the low iron values, indicating anemia of prematurity. In order to correct these problems, the concentration of sodium was reduced in the breast milk and the breast milk was supplemented with iron. Figure 3 demonstrates the weight changes over the first 19 days of life. Weight gain was not steady or significant in this time period, which is common for premature infants.

Baby B:

Gestational Age: 28 ⁵/₇ weeks

Apgars: One minute: 1, Five minutes: 5, 10 minutes: 7

The mother of Baby B had class B diabetes mellitus. The delivery was done via caesarian section. The infant was born experiencing problems with breathing, and as a result was diagnosed with respiratory distress syndrome. There was a possibility of sepsis, which was not ruled out during the seven days of data collection for Baby B, since the infant showed clear signs of sepsis. Hyperbilirubinemia was another problem plaguing Baby B. Hypotension was yet another complication for Baby B. By the sixth day of life, Baby B was also experiencing hypocalcemia. During the seven days of observation, Baby B was NPO. This was routine for the first day of life. By the fifth day of life, the infant was still NPO because residuals were present, indicating feeding intolerance. The medications initially given to Baby B were dopamine and dobutamine to improve heart contractions. After five days, the infant was also given caffeine to stimulate respiration. Due to hyperbilirubinemia, Baby B had to undergo prophylactic phototherapy. Table 3 displays the limited observations recorded for Baby B. There were no significant biochemical markers for Baby B, since data were only recorded for six days. Figure 4 shows the weight changes for Baby B over the six days of observation. Since the observation period was so short, Baby B actually never regained the initial weight loss due to water losses.

Baby C:

Gestational Age: 26 weeks

Length at Birth: 33.5 cm

Apgars: One minute: 6, Five minutes: 7

The mother of Baby C was a 29-year-old woman who delivered by a caesarian section secondary to a breech presentation. The mother was taking Glucophage to improve insulin sensitivity, which was impaired due to her diagnosis of polycystic ovary syndrome. The infant was conceived by intrauterine insemination while the mother was taking Clomid. Clomid is a fertility drug commonly used for women who otherwise cannot become pregnant.

Baby C was a female born at 26 weeks. She was immediately classified as an extremely low birth weight infant, due to the fact that her birth weight was 810 grams. She was diagnosed with respiratory distress syndrome, hyperbilirubinemia, suspected sepsis, and electrolyte imbalances at birth. Phototherapy was initiated to treat the hyperbilirubinemia.

On the second day of life, enteral feeds began for Baby C. She tolerated the feeds with minimal residuals. The energy intake was fair with TPN, but adding enteral feeds with TPN improved the energy intake. Bowel movements did not begin until the fourth day of life, which was the third day on enteral feeds. Residuals disappeared by the fourth day of life. However, weight gain was poor with a fair calorie intake. By the sixth day of life, weight gain began to improve. TPN was discontinued on the fourth day of life as enteral feeds progressed. The enteral formula was composed of breast milk and half-strength PEF 24 at the slow rate of 0.5 ml/hr for the first day of life. PEF 24 is a formula made by Enfamil that contains 24 kilocalories per fluid ounce. It contains docosahexaenoic acid (DHA) and arachidonic acid (ARA), which are essential fatty acids needed for proper eye and brain development (20). A nasogastric route was used to nourish the infant. By the seventh day of life, PEF 24 was used without breast milk to feed the infant. Feeds continually increased throughout the case study, since the infant tolerated the feeds pretty well and experienced promising weight gain.

Baby C was intravenously administered a variety of medications for various conditions. Baby C was given caffeine and Cortef to stimulate respiration. Zantac was used to treat the gastroesophageal reflux that Baby C was experiencing. Gastroesophageal reflux seems to be fairly common in preterm infants. The major problem associated with gastroesophageal reflux is its role in stimulating apnea. While it is not the main cause of apnea, it clearly puts a strain on an infant's respiration (19). In addition, Baby C took Vancomycin as a prophylactic treatment for sepsis. Baby C was given Ferinsol, an iron supplement combined with multivitamins that helps increase limited iron stores. This was supplemented with sodium chloride to treat her hyponatremic condition. Table 4 displays the data that were collected for Baby C, and Figure 5 shows the weight changes for Baby C throughout the case study.

Baby D:

Gestational Age: 27 weeks

Length at Birth: 37 cm

Apgars: One minute: 8, Five minutes: 8

Baby D was born of a 36-year-old mother by a caesarian section secondary to vaginal bleeding. At birth, Baby D was treated with surfactant to ease the difficulty of breathing. The initial problems were hyperbilirubinism, respiratory distress, and PIE. Baby D also had a patent ductus arteriosis (PDA). By day ten of life, this became an important issue. Indocin was used to treat the PDA. It works by closing the ductus.

The nutritional care plan for Baby D began according to the usual protocol. He was NPO on TPN for the first four days of life. On day five, he began enteral feeds with sterile water at a very slow rate of 0.5 ml/hr, using an orogastric route. On day six, Baby D was given breast milk with half-strength PEF 24 formula at the slow rate of 0.5 ml/hr. Since this slow feeding was tolerated, the rate was increased to 2.0 ml/hr on day eight, but on day nine Indocin treatment began. On day 12, Baby D resumed enteral feeds at a rate of 1.0 ml/hr with just breast milk. This only lasted until day 14, when feeding intolerance occurred. On day 15 and 16, enteral feeds were attempted in vain, since they once again resulted in feeding intolerance. On day 20, Baby D resumed enteral feeds at the slow rate of 0.5 ml/hr with breast milk. This progressed until day 26 at a rate of 7.0 ml/hr, to the point that TPN was discontinued completely. Full feeds were well-tolerated, but on day 27 Indocin treatment was implemented once again. Therefore, Baby D had to revert to TPN, but only for nourishment.

Baby D experienced many feeding problems. One recurring feeding problem was associated with the Indocin treatment. Once on Indocin, a patient must be NPO. Each time Baby D was administered Indocin, he was NPO. This occurred at day nine of life and continued through day 11 and again at day 27. Baby D also had to be NPO due to multiple occurrences of feeding intolerance characterized by emesis, residuals, and the absence of bowel movements. Table 5 shows the information that was collected on Baby D, and Figure 6 displays the weight changes for Baby D.

Baby F:

Gestational Age: 30 weeks

Length at Birth: 34 cm

Apgars: One minute: 7, Five minutes: 8

Baby F was a male born of a 21-year-old mother with lupus nephritis and edema. The infant was pale at birth with minor respiratory distress and was found to have hyperbilirubinemia, anemia, and ADP. In addition, Baby F was small for gestational age. These issues did not turn out to be extremely severe. Caffeine was given to Baby F to stimulate respiration. The nutritional care plan for Baby F involved the use of formula and not breast milk. By the third day of life, enteral feeds began for Baby F, with halfstrength PEF 24 at 0.5 ml/hr. Signs of feeding intolerance surfaced on the 11th day of life, when full-strength formula was initiated. To compensate, the rate of enteral feeds was decreased. The infant tolerated the feeds well and at day 14 TPN was discontinued, since feeding intolerance ceased. Baby F progressed as well as could be expected. Feeds were well tolerated and led to significant weight gain. Table 6 shows all of the data that were collected for Baby F during the case study. Figure 7 shows how weight changed throughout the observation period, and Figure 8 displays how the hematocrit changed throughout the case study. Discussion of Case Studies

Tracking five infants does not provide enough information to make generalizations about the nutritional care of premature infants, but it does show that each infant must be handled individually. Each infant is going to have different problems based on when they were born and their stage of development. Just knowing when an infant is born does not tell exact information about the stage of development since each infant develops at their own rate.

Similarities did occur among the infants involving their medical problems. Respiratory distress, hyperbilirubinemia, and anemia were all common issues among the infants. In addition, feeding tolerance and electrolyte balance were difficult to control. The main point is that treating premature infants must be approached in a systematic yet individualized way. Each infant must be examined with knowledge of common signs and symptoms to notice. From that point on, an individualized care plan can be initiated.

Applying the main points to nutritional support leads to some general rules for infant care. Each infant must begin with TPN containing proper nutrients, such as carbohydrates, fat, amino acids, vitamins, minerals, and medications. If, after assessment, the infant has normal bowel sounds and what appears to be a normal gut (i.e. not distended), the infant can be enterally fed sterile water at a very slow rate to test gastrointestinal function. If the water is tolerated, expressed breast milk or preterm formula can be initiated at a slow rate. TPN should still be administered during the early stages of enteral nutrition. If enteral feeds are well tolerated, the rate of feeds can be increased while TPN can be withdrawn until enteral feeds are used alone. Most situations do not allow for the smooth transition from TPN to complete enteral feeds. Feeding intolerance often occurs, slowing the transition process. Certain medications, such as Indomethacin, interfere with the feeding plan. Gastrointestinal problems directly affect how an infant can be fed. If the problems are severe, such as with necrotizing enterocolitis, the infant must be NPO. Other problems, such as respiratory problems, indirectly impact the nutritional care provided to the infant. Most problems increase the energy needs of the infant since the infant must work harder to continue growth and development.

Long-Term Outcomes

Infants born at less than 28 weeks of gestation face the most severe consequences. While these infants account for less than one percent of all infants

born in the United States, their problems require the most attention. They usually are born at very low birth weight and need oxygen, surfactant, and ventilators to allow proper respiration. In addition, most of these infants will have to be fed using TPN. Eighty percent of infants born at 26 weeks will survive to one year of age, while 87 percent born at 27 weeks will survive to one year. However, a significant portion of that year may be spent in the NICU, and the infants face an increased risk of developing permanent disabilities (21).

Infants born between 28 and 31 weeks of gestation are more likely to survive than those born at less than 28 weeks (90 – 95% survive until one year of age), but they still experience many problems at birth. Many of these infants can progress to EN with less difficulty than an infant born at less than 28 weeks. The use of ventilators, surfactant, and oxygen can alleviate breathing problems in these infants. These infants also face the risk of being plagued by lifelong disabilities. Infants born at 32 to 35 weeks of gestation have fewer problems than younger infants. They are unlikely to develop lasting disabilities, and if they have complications at birth, they are usually not as severe as those of younger infants (21).

Since survival rates have increased for preterm infants, it has been possible to examine their long-term outcomes. It was once thought that catch-up growth for weight, length, and head circumference could only occur within the first few years of life, but it now appears that catch-up growth occurs up until age 18. This holds true even for extremely low birth weight infants. However, neurological impairments are more prevalent later in life in infants who were born at low birth weight. Cerebral palsy, cognitive disorders, blindness, autism, and deafness are just some of the neurological impairments present in children born premature (21). Infants born premature are more likely to have neurodevelopmental problems two years after birth than full term infants (22). <u>Discharge Care</u>

The driving factor behind the decision to discharge an infant is feeding status. Preterm infants must have the ability to tolerate all of their feeds and ideally to nipple feed. They also must have the ability to meet their needs for growth on the feeding schedule they will follow at home. In addition, they should be able to thermo regulate without an incubator. Furthermore, any medical problems that the infant has should be able to be adequately managed at home, without any detriment to the infant (23).

Another important part of the discharge process is the preparation of the parents. It takes time for parents to develop enough confidence to take their tiny infant home. Parents also need a great deal of support and education before they can handle being alone at home with the infant (23).

Once the infant does go home, consistent monitoring becomes important, especially in the first month. A nurse and a registered dietitian should make a home visit to the infant within the first week of discharge in an ideal situation (23).

Preterm infants cannot properly feed at home until heart rate is stable, respiratory rate is steady, and muscle tone is adequate. Infants who have little muscle tone and poor muscle control have a difficult time nipple feeding. They tire easily, and feeding can become a laborious chore. Many factors make feeding a preterm infant very difficult after discharge (23).

Final Words

Preterm infants comprise a difficult group for health care professionals to treat. Their high-risk status and multiple medical problems create a situation where each infant must be individually monitored and treated. One, allencompassing nutritional care plan does not exist for these infants. Therefore, each infant's set of conditions must be considered before they can be treated. The limited amount of research that exists on this topic is a direct result of the unethical nature of experimenting with such high-risk individuals. However, the importance of safe, thorough examinations of these infants cannot be emphasized enough. In order to adequately address the nutritional needs of such infants, further examination and assessments must be performed on these infants.

Despite the uncertain nature of caring for these infants after discharge, general guidelines for feeding can be followed. Approximately 100 kcal/kg of body weight is the generally accepted energy intake for premature infants after discharge (24). Since infants who are breast-fed demonstrate signs of reduced growth (13), using formula or a human milk fortifier with expressed breast milk may yield optimal results for growth and development. Breast milk is the preferred form of nourishment with human milk fortifier as directed by the nurses and physicians in the NICU. Formulas containing more than 24 kcal/fl oz tend to create gastrointestinal problems for the infant, including diarrhea and dehydration. If formula must be used, a regular formula should be initiated as opposed to a premature infant formula when the infant reaches the full term age (24). A general rule to follow is to feed the infants every three hours. In some instances, mothers will have difficulty expressing enough breast milk. To correct this problem, the mother should express breast milk every one and a half to two hours for the first two days after discharge. This stimulates the breasts to produce more milk. The mother will know that the infant is adequately fed and hydrated when the infant has six to eight wet diapers each day (24). This usually results from eight to ten feedings each day every two to three hours. Infants should not go without fluid for more than four hours. Solid foods should be introduced until at least four to six months past the infant's due date to allow adequate development of the swallow reflex for solid food. Cow's milk should not be introduced until at least a year after the infant's due date. In addition, infants who are born small for gestational age should not be given solid foods until they have experienced enough catch-up growth to be considered normal for gestational age (24).

Vitamins and minerals may need to be supplemented during this time after discharge. Two to four mg/kg of body weight of iron should be supplemented daily if the infant is using a low-iron formula or breast milk. This supplementation should carry on for 12 to 15 months. In addition, low birth weight infants should be monitored for vitamin D, E, K, and folic acid deficiencies. With the exception of vitamin D, most deficiencies are uncommon. Regardless, vitamin supplements are recommended for breast-fed infants or infants who are not ingesting at least 32 fl oz of formula each day for the first year of life (24). While these guidelines will not be adequate for all infants born premature, they apply to most infants. They provide a starting point for infant care. More specific rules of care for each infant are administered by the professionals in the NICU.

The main goal of nutritional care is to promote normal growth and development. The exact method for doing this is impossible to generalize for all infants due to individual differences, but it is necessary to create basic guidelines. Each infant's case must be reviewed to ensure proper care is given. Examining the nutrition-related problems of premature birth is necessary to advance the techniques used in NICUs throughout the United States, since prematurity is becoming a more prevalent problem. Observing premature infants in a NICU is an effective way to understand the nutritional treatment of these infants in a reallife setting. Taking the time to complete such observations is important because more treatments will be necessary to address this increasing problem of prematurity.

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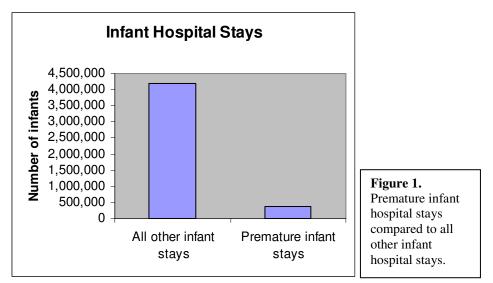
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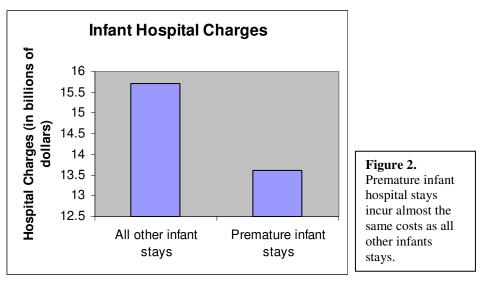
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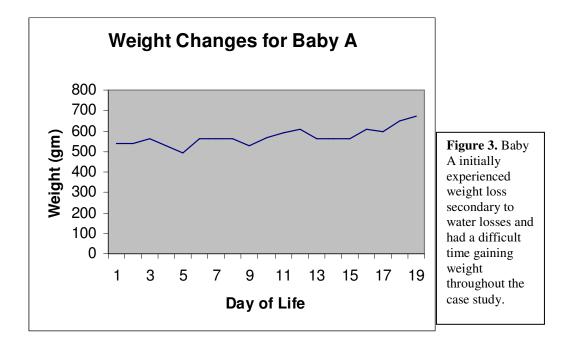
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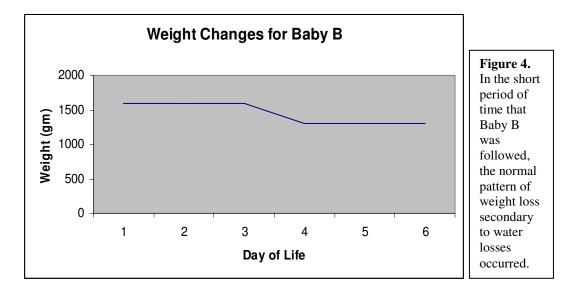


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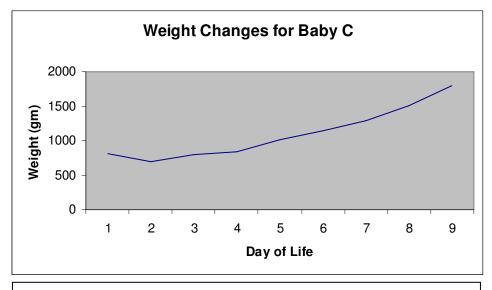
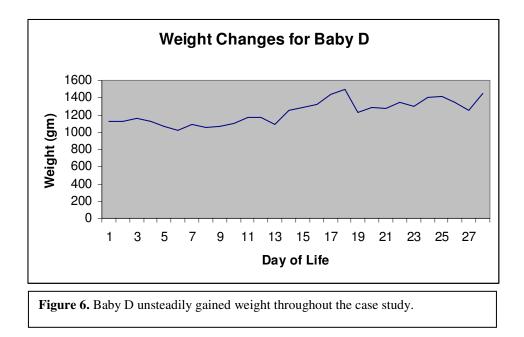
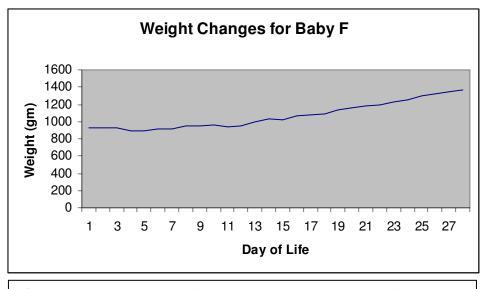
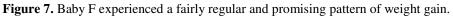
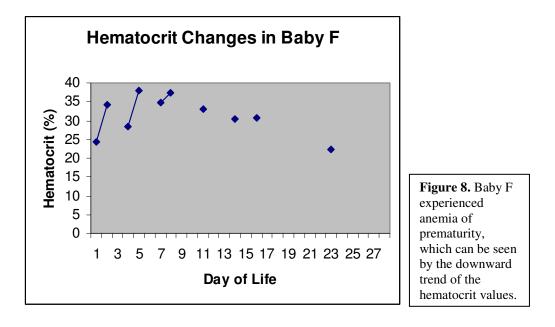


Figure 5. Baby C experienced the initial expected weight loss, but then began to steadily gain weight.









Normal Biochemical Values

Glucose (gm/dl)	70 - 105
BUN (mg/dl)	6 – 22
S. Creatinine (mg/dl)	0.4 - 1.2
Na (MEQ/l)	135 – 145
K (MEQ/l)	3.5 - 5.0
Cl (MEQ/l)	100 - 108
Total Protein (gm/dl)	6.5 - 8.0
S. Albumin (gm/dl)	3.2 - 4.7
T. Bilirubin (gm/dl)	0.2 – 1.3
RBC (M/CU mm)	5.0 - 6.3
WBC (K/CU mm)	5.0-21.0
Differential:	
Segs (%)	19 - 49
Lymphocytes (%)	36 - 46
Monocytes (%)	0 - 11
Eosinophils (%)	0-5
Basophils (%)	0 - 2
Hgb (gm/dl)	18.5 - 21.5
Hct (%)	53.0 - 65.0

Table 1. The normalvalues accepted for eachof the biochemicalparameters for preterminfants.

Baby A: Data and Observations

Day of Life	1	2	3	4	5	6	7
Weight (gm)	540	540	560	530	490	560	560
Head	20.5			19.5	19.5	19.5	19.5
Circumference							
(cm)							
~			hemical				
Glucose (gm/dl)	157	89	133	144	126	61	88
BUN (mg/dl)	13	17	15	14	23	35	42
S. Creatinine (mg/dl)	0.8	1.1	1.1	1.1	1.1	1.1	1.1
Na (MEQ/I)	149	144	149	145	143	136	148
K (MEQ/l)	4.3	5.0	4.4	4.7	4.5	4.5	3.2
Cl (MEQ/l)	114	110	117	113	112	106	113
T. Bilirubin (gm/dl)	3.9	4.4	4.5		3.2	4.7	
RBC (M/CU mm)	2.07	2.53	3.53		3.31	3.69	3.46
WBC (M/CU mm)	18.2	13.8	9.5		11.3	11.1	28.0
Differential:							
Segs (%)		20	33		15	20	19
Lymphs (%)		62	47		61	54	59
Mono(%)		13	13		12	14	12
$\operatorname{Eos}(\%)$		1	3		7	8	4
Baso (%) Hgb (gm/dl)	8.3	8.9	12.0	11.7	1 10.9	11.7	11.1
	0.3	0.9			10.9	11./	11.1
Hct (%)	26.0	27.9	35.5	34.0	33.1	35.2	32.8

Day of Life	8	9	10	11	12	13	14
Weight (gm)	560	530	570	590	610	560	560
Head	19.5	19.5	19	19.5	19.5		20.5
Circumference							
(cm)							
			chemical V	1	1		
Glucose (gm/dl)	102	129	145	81	83	64	81
BUN (mg/dl)	43	35	32	21	15	14	11
S. Creatinine (mg/dl)	1.1	1.2	1.0	1.0	0.9	0.9	0.8
Na (MEQ/l)	148	148	144	141	143	143	143
K (MEQ/l)	3.2	4.7	4.3	5.8	6.4	6.3	5.7
Cl (MEQ/l)	112	114	110	108	112	110	106
T. Bilirubin (gm/dl)	4.9	2.7	2.9	3.6			3.2
RBC (M/CU mm)	3.03	3.48	4.23	4.26			3.94
WBC (M/CU mm)	26.7	39.4	33.5	41.2			39.0
Differential:		16	26	22			96
Segs $(\%)$		16 58	26 58	22 67			86 83
Lymphs (%) Mono (%)		- 38 - 9	- 38 - 10	7			83 5
$\frac{1}{1000} \frac{1}{1000} \frac{1}{1000$		2	3	1			4
Hgb (gm/dl)	9.6	11.2	12.9	13.3		12.5	11.9
Hct (%)	29.2	32.0	38.9	39.3		33.0	36.8

Day of Life	15	16	17	18	19
Weight (gm)	560	610	600	650	670
Head Circumference (cm)	20.5	20.5	20.25	20.25	20.5
	1	Biochemical	Values		
Hgb (gm/dl)	33.2	29	36	31	
Hct (%)	20.5	20.5	20.25	20.25	20.5

Table 2. Data for Baby A collected from day 1 through day 19 of life.

Baby B: Data and Observations

Day of Life	1	2	3	4	5	6	7
Weight (gm)	1590	1590	1590	1310	1300	1300	
		Bioc	hemical `	Values			
Na (MEQ/l)	127.9	133.7					
K (MEQ/l)	5.89	4.01					
Cl (MEQ/l)	96	100					
Hgb (gm/dl)	15.9	11.2	12.4	14.2			
Hct (%)	47	33	43.9	37.3		37	

Table 3. Data collected for Baby B from day 1 through day 6 of life.

Baby (C:	Data	and	Observations
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Day of Life	1	2	3	4	5	6	7
Weight (gm)	810	700	800	835	1010	1140	1285
Head Circumference (cm)	24.5	24.5	23.5	24.5	25.5	26.0	27.5
		Bioc	hemical V	alues			
Glucose (gm/dl)				40	75	69	87
BUN (mg/dl)				10	6	8	3
S. Creatinine (mg/dl)				0.4	0.4	0.3	0.3
Na (MEQ/l)		137	140	133	137	137	141
K (MEQ/l)		4.4	5.1	4.8	5.3	4.4	4.9
Cl (MEQ/l)		103	103	96	104	103	109
RBC (M/CU mm)				3.40	2.75	3.68	
WBC (M/CU mm)				14.8	19.2	13.4	

Day of Life	8	9
Weight (gm)	1505	1795
Head	28.5	29.5
Circumference		
(cm)		
Biocher	nical Valu	es
Glucose	80	
(gm/dl)		
BUN (mg/dl)	5	
S. Creatinine	0.2	
(mg/dl)	0.2	
Na (MEQ/l)	140	
	140	
K (MEQ/l)	5.7	
Cl (MEQ/l)	107	
RBC (M/CU	4.11	
mm)		
WBC (M/CU	10.7	
mm)		
Table 4. Data colled	cted for Baby	C from day 1

Baby D: Data and Observations

Day of Life	1	2	3	4	5	6	7
Weight (gm)	1130	1130	1160	1130	1070	1020	1090
Head Circumference (cm)	25.75				25.25		25.5

Day of Life	8	9	10	11	12	13	14
Weight (gm)	1060	1070	1100	1170	1175	1095	1250
I	25.5			25.0	25.5		
Head Circumference	25.5			25.0	25.5		
(cm)							
(em)		Bioc	hemical V	alues			
Glucose						61	39
(gm/dl)							
BUN (mg/dl)						29	30
S. Creatinine						0.7	0.7
(mg/dl)							
Na (MEQ/l)						132	137
K (MEQ/l)						5.5	5.6
Cl (MEQ/l)						100	101
T. Bilirubin						4.0	3.8
(gm/dl)						2.00	4.00
RBC (M/CU mm)						3.60	4.28
WBC (M/CU						21.9	27.9
mm)						21.7	27.9
Differential:							
Segs (%)						16	16
Lymphs (%)						53	61
Mono (%)						7	8
Eos (%)						10	2
Hgb (gm/dl)						11.0	13.2
Hct (%)						31.5	36.4

Day of Life	15	16	17	18	19	20	21
Weight (gm)	1285	1320	1440	1490	1225	1285	1275
Head Circumference (cm)	26.0		25.5			26.5	26.5
		Bioc	hemical V	alues			
Glucose (gm/dl)	45	120	61	68	70	86	86
BUN (mg/dl)	22	16	33	39	32	31	27
S. Creatinine (mg/dl)	0.6	0.4	0.8	0.6	0.6	0.6	0.6
Na (MEQ/l)	143	135	133	141	141	142	140
K (MEQ/l)	4.3	6.1	5.4	3.9	4.5	3.7	3.2
Cl (MEQ/l)	102	98	95	103	99	96	100
RBC (M/CU mm)	4.02	4.93		3.61	4.75	4.99	4.45
WBC (M/CU mm)	36.8	39.9		46.6	28.1	18.9	10.5
Differential:							
Segs (%)	9	11				16	23
Lymphs (%)	75	73		84		76	63
Mono (%)	7	6		4		4	7
Eos (%)	2	1		9	14.0	2	4
Hgb (gm/dl)	12.3	15.4		10.8	14.2	15.1	12.9
Hct (%)	34.7	42.2		29.9	39.4	41.6	37.8

Day of Life	22	23	24	25	26	27	28
Weight (gm)	1350	1300	1400	1410	1340	1255	1450
Head	26.5		27.0		26.9	27.0	27.0
Circumference (cm)							
(em)		Bioc	hemical V	alues			
Glucose (gm/dl)	65	92	43		52		
BUN (mg/dl)	25	27	21		12		
S. Creatinine (mg/dl)	0.5	0.5	0.4		0.4		
Na (MEQ/l)	138	136	138		142		
K (MEQ/l)	4.4	4.2	4.6		5.0		
Cl (MEQ/l)	99	97	99		98		
RBC (M/CU mm)	4.36			3.78	5.23		
WBC (M/CU mm)	16.0			11.1	13.6		
Differential:	10			10			
Segs $(\%)$	12			42	26		
Lymphs (%) Mono (%)	77 7			39 12	48 20		
$\frac{1}{1000} \frac{1}{1000} \frac{1}{1000$	3			2	20 4		
Baso (%)				1			
Hgb (gm/dl)	12.9			10.7	13.8	15.1	
Hct (%)	37.3			31.6	41.3	44.0	
						I	

Table 5. Data collected for Baby D from day 1 through day 28 of life.

Baby F: Data and Observations

Day of Life	1	2	3	4	5	6	7
Weight (gm)	930	930	930	890	890	920	920
Head Circumference (cm)	25.5		25.5			25.0	25.0
		Bioc	hemical V	Values			
Glucose (gm/dl)		95				95	72
BUN (mg/dl)		26				5	5
S. Creatinine (mg/dl)		1.3				0.7	0.7
Na (MEQ/l)		139	142	141	140	138	141
K (MEQ/l)		5.2	5.1	4.6	5.0	4.1	4.6
Cl (MEQ/l)		105	111	114	111	112	111
T. Bilirubin (gm/dl)			6.7	7.8	7.9	7.3	5.8
RBC (M/CU mm)	2.46	3.89		3.07	4.49		4.13
WBC (M/CU mm)	5.1	9.2		7.9	11.3		11.0
Differential: Segs (%) Lymphs (%) Mono (%) Eos (%)	22 67 9 1	59 34 6		53 35 5 7	37 42 19 2		40 36 20 4
Hgb (gm/dl)	7.9	12.0		9.4	13.8		12.7
Hct (%)	24.4	34.3		28.4	37.9		34.8

Day of Life	8	9	10	11	12	13	14
Weight (gm)	956	950	960	935	955	995	1030
Head Circumference (cm)	25.5		25.5	25.5	26.0	26.0	25.0
		Bioc	hemical V	Values			
Glucose (gm/dl)			65			67	73
BUN (mg/dl)			7			3	4
S. Creatinine (mg/dl)			0.6			0.5	0.5
Na (MEQ/l)	138	135	138	138	140	137	140
K (MEQ/l)	6.0	5.7	5.8		4.7	5.1	4.6
Cl (MEQ/l)	108	107	108		110	105	108
T. Bilirubin (gm/dl)	5.3	5.1	5.1	6.1			
RBC (M/CU mm)	4.33			3.76			3.42
WBC (M/CU mm)	10.8			11.4			10.3
Differential: Segs (%)				24			25
Lymphs (%)				53			55
Mono (%)				21			18
Eos (%)				2			2
Hgb (gm/dl)	13.1			11.1			9.9
Hct (%)	37.4			33.1			30.4

Day of Life	15	16	17	18	19	20	21
Weight (gm)	1020	1065	1080	1085	1140	1160	1185
Head	25.0	25.0	26.0	26.0		27.0	27.0
Circumference (cm)							
(CIII)		Bioc	hemical V	/alues			
Glucose		69		aiues			
(gm/dl)							
BUN (mg/dl)		3					
S. Creatinine		0.4					
(mg/dl)		142		1.4.2			
Na (MEQ/l)		143		143			
K (MEQ/l)		4.9					
Cl (MEQ/l)		109					
Total Protein		4.7					
(gm/dl)				1.0	4.1		1.0
T. Bilirubin (gm/dl)		7.6		4.2	4.1		4.8
RBC (M/CU		3.45					
mm)		5.45					
WBC (M/CU		11.7					
mm)							
Differential:							
Segs (%)		32					
Lymphs $(\%)$		42 22					
Mono (%) Eos (%)		3					
Hgb (gm/dl)		9.9					
Hct (%)		30.8					

Day of Life	22	23	24	25	26	27	28
Weight (gm)	1200	1230	1250	1300	1320	1345	1370
Head Circumference (cm)				27.5	28.0		
			hemical V	alues			
Glucose (gm/dl)		99					
BUN (mg/dl)		2					
S. Creatinine (mg/dl)		0.4					
Na (MEQ/l)		138			138		
K (MEQ/l)		4.5					
Cl (MEQ/l)		106					
Total Protein (gm/dl)		4.5					
S. Albumin (gm/dl)		3.2					
T. Bilirubin (gm/dl)		4.8					
RBC (M/CU mm)		2.58					
WBC (M/CU mm)		9.1					
Differential:							
Segs (%)	——	21					
Lymphs (%)	——	65					
Mono (%) Equation (%)		10 4					
Eos (%) Hgb (gm/dl)		7.3					
		,					
Hct (%)		22.3					

Table 6. Data collected for Baby F from day 1 through 28 of life.