Nutrition in Paediatric Patients

Module 4.4

Principles of Feeding the Preterm Infant

Koen Joosten
associate Professor Pediatrics
MD, PhD, Pediatric Intensivist
ErasmusMC-Sophia Children’s Hospital Rotterdam
The Netherlands

Learning Objectives

- Understanding the differences in pre- and postnatal growth factors.
- The importance and impact of early aggressive nutritional support.
- Timing of nutritional support concerning start of parenteral and enteral nutrition.
- Supplementation of vitamins and micronutrients.
- Treatment of parenteral nutrition-associated liver disease.

Contents

1. Introduction and rationale of feeding the preterm infant
   1.1 General
   1.2 Prenatal and postnatal growth factors
   1.3 Postnatal growth failure
2. Nutritional management for the preterm infant
   2.1 Fetal nutrition
   2.2 Early parenteral nutrition
      2.2.1 Fluids
      2.2.2 Carbohydrates
      2.2.3 Proteins/Amino acids
      2.2.4 Lipids
      2.2.5 Calcium, phosphorus and Vitamin D
      2.2.6 Iron
      2.2.7 Pre- and probiotics
   2.3 Early enteral nutrition
      2.3.1 Trophic or minimal-enteral feeding
      2.3.2 Choice of milk
3. Post-discharge feeding
4. Summary
5. References

Key Messages (5-6)

- Human milk is preferred in preterm infants for its protective effects against necrotising enterocolitis, infection and neurodevelopmental delay. As breast
milk lacks sufficient protein and energy for optimal growth and development of the preterm infant breast milk fortifier should be added.

- Very low birth weight infants are prone to growth restriction and altered body composition. Early parenteral protein and lipid administration can prevent protein loss and facilitates growth.
- The in-hospital postnatal growth rate of preterm infants should approach fetal growth, in quantity and in quality, so that both size and body composition of a preterm infant at term-corrected age are equal to those of the term-born infant.
- Growth is essential for preterm infants after discharge from the hospital and (fortified) breast milk or post-discharge formula should be given until a SD score of -1 is reached or until 6 months of age (corrected for prematurity).
- Amino acids are pivotal in early life for synthesis of proteins, neurotransmitters, growth factors and other biologically active molecules. When receiving only glucose after birth, the estimated protein loss is approximately 1% of the endogenous body protein per day.
- Both AA and DHA should be provided because these polyunsaturated fatty acids have beneficial effects on cognitive development.

1. Introduction and Rationale of Feeding the Preterm Infant

1.1 General

Preterm birth, defined as being born before 37 completed weeks of gestation, is the leading cause of perinatal mortality and morbidity in developed countries. Major innovations, such as artificial ventilation and antenatal steroids, have increased the survival rates significantly. However, morbidity rates, e.g. of growth failure and long-term neurodevelopmental impairment, are still high, and of great concern for neonatologists (1). Low in-hospital growth velocity is associated with cerebral palsy, MDI and PDI scores of <70, and neurodevelopmental impairment (2).

It is recommended by the American Academy of Pediatrics that the postnatal growth rate of preterm infants duplicates fetal growth, in quantity and in quality, so that both size and body composition of a preterm infant at term-corrected age are equal to those of the term-born infant. Over the last decades, dramatic improvements in neonatal medicine have resulted in regular survival of prematurely born infants as young as 24 weeks’ gestational age and as small as 500 g. Nowadays, these very small, immature infants are relatively common in neonatal intensive care units and a challenge is to achieve healthy growth over a 12-16 week postnatal period. Although over the last decades much progress has been made in the field of nutrition, many uncertainties remain about the requirements of preterm infants. Short- and long-term effects of inadequate feeding and hampered growth are increasingly recognized. Still, many premature infants remain significantly growth restricted at hospital discharge (3).

1.2 Prenatal and Postnatal Growth Factors

During infancy and childhood, growth is growth hormone (GH)-dependent, while prenatal growth is not. Although GH is high during pregnancy, GH has little effect on the fetal insulin-like growth factor (IGF) axis. Both animal studies and human studies show that insulin like growth factor type II (IGF-II) is the dominant factor for fetal
growth. The role of IGF-II diminishes during pregnancy, with no influence on growth at the end of the third trimester. Of interest is that early fetal growth is independent of nutrient supply, since the influence of nutrition on IGF-II is limited. Insulin growth factor type I (IGF-I) is taking over the role as growth regulator from IGF-II during the second half of pregnancy. IGF-I production and secretion by the liver is influenced by thyroid hormones, glucocorticoids, nutrient supply and insulin secretion. In contrast to the first part of pregnancy, nutrient supply influences growth widely during the second part of pregnancy.

In healthy term-born infants fetal growth mechanisms remain dominant after birth for the first 6 months. Thereafter, there is a shift from IGF-I dependency (which allows influence by the environment, e.g. by nutrition) to IGF-I/GH dependency (no influence of nutrition, thereby it is possible to gain maximal growth potential).

Little is known about the timing of this shift in preterm infants. Therefore, it is not known if preterm infants are able to grow adequately in the extra uterine third trimester.

### 1.3 Postnatal Growth Failure

Growth failure in preterm infants is common, and results from a complex interaction of many factors, including morbidities affecting nutrient requirements, endocrine abnormalities, central nervous system damage, difficulties in suck and swallow coordination, and administration of drugs that affect nutrient metabolism. Furthermore, prematurity, intrauterine growth retardation (IUGR) and extra-uterine growth retardation (EUGR) are associated with long-term metabolic and cardiovascular consequences. However, the principal contributor to postnatal growth restriction during hospital stay is malnutrition, often due to inadequate provision of nutrients because of fear of intolerance of parenteral nutrition (4).

Extra-uterine growth retardation (EUGR) is most often defined as weight, length or head circumference below the 10th percentile at discharge. The incidence of EUGR varies between 43%–97% in various centres. The incidence of EUGR increases with decreasing gestational age and birth weight. Factors that are independently associated with EUGR are male gender, need for assisted ventilation on day 1 of life, a history of necrotizing enterocolitis, oxygen dependency at 28 days of age and the need for steroid use during the hospital stay (5).

The current feeding strategy for preterm infants includes early, aggressive parenteral nutrition, containing carbohydrates, proteins and lipids. Several studies have shown that preterm infants, despite the aggressive feeding strategies nowadays, are significantly lighter and shorter and have an altered body composition when reaching corrected term age than those who are born at term; they have a higher total fat mass at term corrected age, caused by significantly less fat-free mass but a more similar fat mass to that of healthy term-born infants, with a distribution in favour of intra-abdominal regions.
2. Nutritional Management of the Preterm Infant

2.1 Fetal Nutrition

During human pregnancy, the fetus receives nutrients via the umbilical cord. Besides glucose, used as a fuel, the fetus receives amino acids as well. In utero, the transfer of glucose across the placenta averages 8 mg/kg/min in the third trimester. Amino acids are also transferred across the placenta continuously. This transfer of amino acids greatly exceeds fetal protein accretion requirements. The amino acid excess is oxidized, thereby contributing significantly to fetal energy generation. Lipids are transported to the fetus in the form of long chain polyunsaturated fatty acids (PUFA). Although PUFAs are transported throughout gestation to the fetus, more than 90% of fetal fat mass is deposited during the third trimester. Lipids are of particular relevance because of their numerous physiological functions (metabolic, energetic and structural), and for the supply of essential n-3 and n-6 fatty acids, which are necessary for the development of the central nervous system of the fetus.

2.2 Early Parenteral Nutrition

Because of the inability of most preterm infants to tolerate full enteral feeding due to immaturity of the gastro-intestinal tract, parenteral nutrition (PN) is needed to feed these infants. However, while PN is life-saving, it is also associated with increased risk of sepsis from cathether-associated infections and progressive liver dysfunction from prolonged parental lipid use. The sudden change from the well-fed state in utero to the extra-uterine environment challenges the preterm infant.

Both growth and disease demand high caloric and amino acid intake. A preterm infant of 1 kg consists of only 1% fat and 8% protein and has a non-protein caloric reserve of 110 kcal/kg body weight. When preterm infants do not receive exogenous substrates after birth, either enteral or parenteral, the infant reaches a catabolic state immediately. When receiving only glucose after birth, the estimated protein loss is approximately 1% of the endogenous body protein per day. The resulting protein deficit may be difficult if not impossible to recoup, and thereby hampers the infants' growth and neurodevelopment. It is therefore of importance that PN is started immediately after birth (6).

2.2.1 Fluids

Fluid tolerance is limited in the first days of life due to renal adjustment but there is large variability among very low birth weight infants. Commonly fluid intake is increased daily in the first week of life. Fluid volumes between 80 and 200 ml/kg/day are tolerated well and these values may serve as lower and upper limits. A postnatal intake at the lower end of the range is likely to minimise risk of long-term morbidity such as brochopulmonary dysplasia and patent ductus arteriosus. Fluid restriction is limited by the osmolarity of parenteral and enteral nutrition that can be tolerated. Current guidelines recommend 135 ml/kg/day as the minimum fluid volume and 200 ml/kg/day as a reasonable upper limit, to be reached after several days.
2.2.1 Carbohydrates

Carbohydrates are a major source of energy. Glucose is the principal circulating carbohydrate, the primary source of energy for the brain and the only carbohydrate in PN. The balance between supply and consumption of glucose determines the plasma glucose levels. The supply is partly exogenous, by enteral or parenteral nutrition and partly derived from endogenous production, e.g. gluconeogenesis and glycogenolysis. Glucose can be used in cells for energy generation or it can be stored, in the form of fat or glycogen. In utero the transfer of glucose across the placenta averages 8 mg/kg/min. In premature infants, the endogenous glucose production is not adequate to provide the demands and, there are few alternative fuels. Therefore, premature infants are dependent on parenteral glucose administration. Furthermore, the insulin response to hyperglycaemia is limited in VLBW and, especially in SFGA babies, insulin resistance is more pronounced. Lipid emulsions may however help to stabilise glycaemia as glycerol is a substrate for gluconeogenesis.

Current guidelines suggest starting with a glucose infusion at 6 mg/kg/min directly after birth, with a daily increase rate of 1-2 mg/kg/min, or more if hypoglycaemia (<50 mg/dl or 2.7 mmol/L) occurs. Generally the maximal glucose intake is 12 mg/kg/min. If blood glucose exceeds 8 mmol/L glucose administration can be decreased by 2 mg/kg/min but not below 6 mg/kg/min. Consider insulin infusion at a rate of 0.01-0.04 IU/kg/hr depending on the blood glucose level and increase insulin up to 0.2 IU/kg/hr to keep the blood glucose at 3-8 mmol/L (7, 8, 9).

2.2.2 Proteins/Amino Acids

Amino acids are pivotal in early life as precursors for proteins (and thus growth) and neurotransmitters, as transport molecules and in cell signalling. Each amino acid has its unique function. Amino acids are classified as essential or non-essential, depending on whether they can be derived only from the diet (essential), or whether they can also be produced endogenously from other substrates in sufficient amounts (non-essential). Of the 20 amino acids present in protein, 9 are essential in human adults. However, due to immaturity of different enzyme systems, premature infants are not able to synthesize an additional 4 amino acids, namely arginine, glycine, proline and tyrosine (conditionally essential amino acids).

Amino acids are continuously synthesized and broken down for protein synthesis and gluconeogenesis. There is an inverse relation between post-conceptional age and the speed of this process. Due to the fixed sequence of amino acids in all proteins, the rate of protein synthesis will be determined by the first limiting amino acid pool. When an essential amino acid is deficient protein synthesis stops and proteolytic rates increase.

A significant part of the amino acids that are released by protein breakdown is reused for protein synthesis. However, a part of it will be lost by renal excretion. This loss is approximately 0.6-1.1 gram/kg/day in preterm infants. Since protein synthesis is an energy-demanding process, sufficient non-protein caloric intake should be administered as well.

During the last decades the beneficial effects of starting amino acid administration directly after birth have been shown. This can reverse a negative nitrogen balance, and this indicates protein synthesis and thus growth. Furthermore, early amino acid
administration increases plasma amino acid concentrations towards ranges found in fetuses and healthy term newborns.

Early amino acid administration will generally result in improved growth at 36 weeks postmenstrual age or at hospital discharge, although this is not consistently reported in all studies. Furthermore, retrospective analyses showed that an increase of 1 g/kg/day of protein intake during the first week of life is associated with an 8-point increase in mental developmental index (Bayley Scales of Infant Development) at 18-22 months corrected age.

Not only the timing and quantity, but also the quality (composition) of the amino acid solution is of great importance for achieving optimal growth and development. Tolerance of amino acid infusions is commonly measured by plasma urea concentrations and ammonia concentrations. It is important to mention that increased urea concentrations are not only a sign of intolerance, but often reflect dehydration or insufficient supply of essential amino acids or calories and therefore amino acid oxidation.

Current guidelines recommend starting amino acid supply on the first postnatal day, with an amount of 2.4 g/kg/day. A further increase to 4.0-4.5 g/kg/day for infants up to 1000g and 3.5 – 4.0 g for infants from 1000 to 1800 g is recommended. The amino acid intake can be reduced towards discharge if the infant’s growth pattern allows for this (8, 10).

**2.2.3 Lipids**

Dietary lipids provide the preterm infant with much of its energy needs, essential polyunsaturated fatty acids, and lipid soluble vitamins. The amount and composition of dietary lipids affect both growth pattern and body composition. The availability and metabolism of long-chain polyunsaturated fatty acids have direct implications for cell membrane functions and the formation of bioactive eicosanoids. Brain grey matter and the retina are particularly rich in long-chain polyunsaturated fatty acids, and complex neural functions are related to energy supply and the composition of dietary fatty acids. Parenteral lipid emulsions are an attractive source of nutrition, because of their high energy density (9 kcal/g) – compared to glucose (4 kcal/g). These extra calories can be used for the high-energy cost of protein synthesis. The high energy density is furthermore useful since fluid restriction is commonly necessary in preterm infants.

Parenteral lipid emulsions can contain both long- and medium-chain triglycerides. The physical, chemical and metabolic properties of triglycerides are determined by their fatty acid contents. Saturated, monounsaturated and polyunsaturated fatty acids differ in their metabolic and physiological properties. While saturated fatty acids serve primarily as an energy source, polyunsaturated fatty acids play an important role as components of structural lipids, for example in biological membranes. Polyunsaturated fatty acids of the n-6 series (linoleic acid (LA) and metabolites), and polyunsaturated fatty acids of the n-3 series (α-linolenic acid (ALA) and metabolites) cannot be synthesized de novo by higher organisms and are, thus, essential nutrients. Furthermore, in premature infants there is a limited capacity to synthesize arachidonic acid (AA) and docosahexaenoic acid (DHA) from linoleic acid (LA) and α-linolenic acid (ALA) and therefore these fatty acids are also considered essential. Clinical trials in preterm infants fed formulae containing both AA and DHA have shown beneficial effects on the developing visual system and measures of cognitive development during the first year of life.
A major concern during administration of parenteral lipids is the development of parenteral nutrition associated liver disease (PNALD). PNALD represents a spectrum of symptoms varying from mild cholestasis to end stage liver disease requiring liver transplantation. The prevalence of PNALD in infants differs widely, ranging from 15-85%. The most significant risk factor is prematurity. This may be due to the reduced bile acid pool size and immature enterohepatic circulation in preterm infants. Furthermore, preterm infants are more likely to be in need of long term PN.

Concerning the use of parenteral lipids there has been a development over the last decades in the type of lipid emulsions. The purely soybean oil-based lipid emulsions are considered to be the first generation lipids and are still the most widely used. Soybean oil is very rich in n-6 polyunsaturated fatty acids (PUFA), at around 60% of the total fatty acid content. Nowadays, soybean oil is looked at with concerns, mainly because of the excess of n-6 PUFAs that can lead to increased oxidative stress. The mechanism by which soybean oil emulsions contribute to the development of PNALD depends on the high level of phytosterols and n-6 PUFAs in soybean oil.

Second generation lipid emulsions are a mixture of soybean oil and coconut or olive oil, delivering medium-chain triglycerides. Hereby, the amount of n-6 PUFA is decreased; potentially reducing the inflammatory consequences such as PNALD. Third generation lipid mixtures typically contain soybean oil for the supply of essential fatty acids, coconut for the delivery of medium-chain triglycerides for rapid provision of energy, olive oil to reduce the n6-n3 ratio and to supply vitamin E, and fish oil for its anti-inflammatory effects. Beneficial effects of these 4-component mixtures on liver function have been described both in adults as in children, but not yet in preterm infants. However beneficial effects on growth and infection rates are seen in preterm infants. In a recent meta-analysis, mixed lipid emulsions were associated with a 25% reduction in sepsis episodes compared with pure soybean oil emulsions. At this moment, fish oil is increasingly gaining favour for the treatment of PNALD, although outcome studies are limited (11, 12, 13, 14).

The current recommendation is to start intravenous lipid administration not later than on the third day of life, but it may be started on the first day. The maximum dose of parenteral lipid administration is 3-4 g/kg/day, which dose can be reached within 3 days from starting. In order to prevent essential fatty acid deficiency, 0.25 g/kg per day linoleic acid (ALA) should be included.

Concerning enteral lipid intake to meet energy needs, an intake of 4.8 to 6.6 g/kg/day or 4.4 to 6.0 g/100 kcal (40-55 En%) is recommended. From the total delivered energy a minimum of 4.5% energy should be delivered from LA and 0.5% from ALA. Recommended intakes are: for DHA 12 to 30 mg/kg/day; and for AA 18 to 42 mg/kg/day (ratio DHA-AA 1:2). The tolerance of lipids can be checked by determining plasma triglyceride and cholesterol levels.

### 2.2.4 Calcium, Phosphorus and Vitamin D

The total amounts of calcium and phosphorus accreted in fetal life are inversely correlated with body weight. In preterm infants the retention of Ca and P is proportional to growth. To ensure appropriate mineralisation of bone in very low birth weight infants and to diminish the risk of fractures and clinical symptoms of osteopenia supplementation of sufficient amounts of calcium, phosphorus and vitamin D is necessary.
Calcium absorption depends on calcium and vitamin D intakes, and calcium retention is additionally related to absorbed phosphorus. The calcium to phosphorus ratio may be an important determinant of calcium absorption and retention. The present recommendation for preterm formula is a calcium to phosphorus ratio close to 2:1. Vitamin D is important for supporting a large number of physiological processes such as neuromuscular function and bone mineralisation. The pathways of Vitamin D absorption and metabolism are fully operative in premature infants from about 28 weeks of gestation.

Current guidelines recommend a parenteral intake of calcium of 1.3 -3.0 mmol/kg/day (52-120 mg/kg/day) and 1-2.3 mmol/kg/day phosphorus (30-70 mg/kg/day). Because only 50-65% of enteral delivered calcium is absorbed, a higher enteral intake of calcium is recommended, from 3-3.5 mmol/kg/day (120-140 mg/kg/day). In addition the amount of enteral phosphate should be 2.1–3 mmol/kg day (60 to 90 mg/kg/day). Individual needs can be determined by measuring spot urinary calcium and phosphate excretion.

Considering the high prevalence of vitamin D deficiency is pregnant women a higher enteral vitamin D supply in preterm infants is recommended; premature infants <1250 gram 1000 IU/day and >1250 gram 800 IU/day (7).

### 2.2.5 Iron

Iron is an essential micronutrient that plays a critical role in many cellular functions and processes, including growth and brain development. Premature infants are especially susceptible to iron deficiency anaemia because of their smaller iron store and greater iron requirements compared to term infants. Human milk and standard formula feeds contain insufficient iron for the needs of premature infants. There is strong evidence that iron deficiency leads to long-term injury of cognitive and motor development. On the other hand, iron overload is harmful for the liver, the immune system and the brain. Furthermore, iron is a pro-oxidant, and non-protein bound iron has been suggested to cause free oxygen radicals and thereby an increase in retinopathy of prematurity. Excess iron supplementation has been shown to increase the risk of infections, delay psychomotor development and decrease length growth. Thus, one must prevent not only iron deficiency but also iron overload (15).

Current guidelines recommend starting iron supplementation 2-6 weeks after birth with an intake of 2-3 mg/kg/day. Supplementation should be continued until the age of 6-12 months, depending on diet.

### 2.2.6 Pre- and Probiotics

**Prebiotics**

Human milk contains more than 130 different oligosaccharides that are fermented in the term infant’s colon. Preterm infants show some absorption of intact human milk oligosaccharides, but most resist digestion in the small intestine and undergo fermentation in the colon. The composition of oligosaccharides in human milk is genetically determined, explaining the large variability in oligosaccharide composition which exists. Therefore, it is difficult to define the exact or ideal oligosaccharide composition of human milk. One type of oligosaccharide mixture (GosFos) has been systematically studied in formula feeds for term and preterm infants. It has been hypothesized that GosFos may accelerate feeding advancement, reduce the...
incidence of gastrointestinal complications such as necrotizing enterocolitis, improve immunological functions, reduce the incidence of hospital-acquired infections, and improve long-term outcome, but there are no data available from studies in preterm babies to support these assumptions (16).

**Probiotics**

Probiotics are live microbial supplements that colonize the gut while providing benefits to the host. The benefits include an improved gut barrier, enhanced mucosal IgA responses, and increased production of anti-inflammatory cytokines hereby reducing the incidence of necrotising enterocolitis (NEC). In a recent meta-analysis of trial data, enteral probiotic supplementation significantly reduced the incidence of severe NEC (stage II or more) and mortality. There was no significant reduction of nosocomial sepsis. The included trials reported no negative side effects and no systemic infection with the supplemented probiotic organisms. Probiotic preparations containing either lactobacillus alone or in combination with bifidobacterium were found to be effective. The authors conclude that head to head comparative studies are required to assess the most effective probiotic strains and combinations, timing, and length of therapy (17).

Current guidelines maintain that there is not enough evidence to recommend the routine use of probiotics or prebiotics in preterm infants. This updated review however strongly supports a change in practice.

**2.3 Early Enteral Nutrition**

**2.3.1 Trophic or Minimal-ental Feeding**

At birth, the gastro-intestinal tract of the premature infant is immature, both morphologically and functionally. Motility is sparse, barrier function is incomplete and immunological defence is immature. Due to the hospital environment and antibiotic use, the prevailing microbiota are abnormal. All of these factors predispose to necrotizing enterocolitis (NEC). Because gut hormone secretion and motility are stimulated by ingesting milk, delayed enteral feeding could diminish functional adaption of the gastrointestinal tract, and result in later feeding intolerance. Lack of enteral nutrition causes gut atrophy, with an increased risk of bacterial translocation. Animal studies have shown that gut growth and functional enzyme activity can be expected from enteral feeding of as little as 30% of daily requirements. Infants given trophic feeding or minimal enteral feeding (MEF) show enhanced activity of digestive enzymes, increased digestive hormone levels and improved gut motility compared to infants not receiving MEF. It has been shown that MEF is tolerated well in preterm infants, and is associated with earlier achievement of full enteral feeding, decreased duration of parenteral feeding and decreased length of hospital stay, without an increase in the incidence of NEC. Therefore, it is of importance to provide preterm infants with small volumes of milk during the first week of life to promote intestinal maturation. MEF can be defined as the administration of very small amounts of enteral feeding, which may be less than 25 ml per day to infants who are still dependent on parenteral feeding. MEF does not significantly add to the nutritional intake, as this small amount of nutrients is largely consumed in the gut (18, 19).
Current guidelines recommend starting MEF on the day of birth in infants who are not able to receive normal enteral feeding. There is a strong preference for milk from the child's mother, 12 to 25 ml each day, to be divided into 6-12 portions. If MEF is well tolerated enteral feeding can be increased.

2.3.2 Choice of Milk

Human milk has a number of benefits for preterm infants. It provides antibodies, enzymes, probiotics, hormones and growth factors. Enteral feeding has three main functions in preterm infants; (1) provision of nutrients, (2) mechanical and biochemical activation of the gut and (3) immunomodulation. Moreover, human milk seems to reduce the incidence of several adverse outcomes, such as necrotizing enterocolitis (NEC), late onset sepsis, retinopathy of prematurity and abnormal brain development. Besides, infants fed their own mothers’ milk are known to tolerate full enteral feeding earlier than their formula-fed peers.

However human milk lacks sufficient protein and energy for optimal growth and development of the preterm infant. Caloric and macronutrient content differ widely between mothers and these can be influenced by several factors, such as stage of lactation, frequency of breast feeding, time of the day, parity, maternal diet and age. As the protein content of breast milk and the neonate's need for protein vary, 'individualised' or 'targeted' fortification has been proposed.

Mothers of preterm infants often have insufficient milk supply in the first postpartum days. Maternal illness, the technique of pumping milk, maternal medication and stress can hamper lactation. Lactational support is often required. Nowadays, in most neonatal intensive care units banked donor milk is supplied. The safety of donor milk can be guaranteed if selection of donors and pasteurisation of milk is protocolized. Although, pasteurisation, freezing and thawing of milk does reduce the bioactivity of several human milk components, donor milk is nonetheless considered the optimal alternative for mother's own milk.

Current guidelines recommend adding breast milk fortifier to meet the infant's nutritional requirements; this contains extra protein, energy, vitamins and minerals. Fortifiers contain approximately 0.8 g of protein per 100 ml of milk. Fortification can be started when 100 ml/kg of enteral feeding is tolerated.

3. Post-discharge Feeding

Preterm infants remain smaller and have suboptimal bone mineral mass throughout infancy, and even during adulthood compared to term-born infants. Therefore, after discharge, a sufficient amount of nutrients is needed to achieve or continue optimal growth and development. Catch-up growth and obesity early in life is associated with metabolic syndrome in later life. Continued growth monitoring is recommended to adapt feeding to individual requirements and to prevent under- or overfeeding and its negative consequences later in life. Human milk, supplemented with fortifier, is preferred for premature infant feeding. If human milk is lacking, a formula feed designed for preterm infants is the second best option. Standard formula feeds are designed for term infants, and based on the composition of mature breast milk. They are relatively low in calories, and contain too little protein for optimal brain development and growth of preterm infants (20).
Current guidelines recommend special post-discharge feeding for preterm infants until an SD score of -1 is reached and for no longer than 6 months after term.

4. Summary

Nutritional support of the VLBW infant should be started as early as possible, not only for growth but also for optimal neurocognitive development. The in-hospital postnatal growth rate of preterm infants should approach fetal growth. Nutrition may need to include total parenteral nutrition in the first few days after birth as intestinal function is immature. The parenteral nutrition should supply the full requirements and contain a generous provision of amino acids. It is recommended to start with minimal enteral feeding on the day of birth in infants who are not able to receive normal enteral feeding. There is a strong preference for milk from the child's own mother. There is an extra need for vitamin D and iron, and supplementation is often necessary.

5. References


