Preterm Infant Nutrition
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Enteral Nutrition in the Preterm Infant. Schanler RJ. Available at: www.uptodateonline.com


With the improved survival rate of very low-birthweight (VLBW) (<1,500 g) and extremely low-birthweight (ELBW) (<1,000 g) infants, pediatricians are managing the nutritional needs of an increasing number of vulnerable neonates. The initial management of preterm infants depends on their weight, gestational age, and severity of illness. There is great institutional variability in neonatal intensive care unit (NICU) feeding protocols.

ELBW/VLBW infants should receive early aggressive nutrition. On day 1, infants often are begun on parenteral nutrition with glucose, protein, and electrolytes. Trophic enteral feedings of human milk or half- to full-strength preterm formula are begun oncepressor agents are discontinued. Initiating trophic feedings early shortens the time to full feedings, but does not decrease the risk of necrotizing enterocolitis (NEC).

As enteral feedings are advanced, hospitalized preterm infants receive supplemented human milk (preferentially) or preterm formula. Compared with term formula, some constituents unique to preterm human milk include a lipid profile that increases fat absorption, a predominance of whey proteins that improves digestion, and maternal antibodies to improve immunity.

Human milk (and supplemented preterm formula) contains "conditionally essential" amino acids (taurine, cysteine, and glutamine). Older infants can synthesize these amino acids, but preterm infants, because of their immature metabolic pathways, are unable to produce them endogenously. These amino acids improve fat digestion, brain and retinal development, and immune function. Glutamine (which may improve gut motility and immunity) and sometimes cysteine are the only conditionally essential amino acids not added to parenteral nutrition.

All enteral nutrition for VLBW/ELBW infants needs to be supplemented. Human milk is supplemented with human milk fortifier to prevent hyponatremia, hypoproteinemia, rickets, and zinc deficiency. Preterm formula is supplemented with electrolytes, calcium, phosphorous, other minerals, water, and fat-soluble vitamins, medium-chain triglycerides, taurine, and calories (24 kcal/oz). Both preterm and term formulas have a predominance of whey.

Preterm infants should not be fed soy formula because of its inadequate calcium/phosphorous content, which can elevate the risk for developing osteopenia and rickets. The NICU closely monitors the infant's growth and nutritional laboratory findings (alkaline phosphatase, calcium/phosphorous, and albumin or prealbumin).

Clinicians have a crucial role in promoting and supporting human milk as the optimal nutrition for preterm infants. Human milk decreases the risk of NEC 6- to 10-fold, shortens the time to full feedings, increases developmental scores, decreases infection rates, and improves maternal-infant bonding. In addition, once the infant is taking oral feedings, nursing on the breast is more physiologically stabilizing with regard to heart rate, respiratory rate, and oxygenation than is bottle-feeding. Mothers should be encouraged to start breast pumping as soon as possible after birth, using a hospital-grade double electric pump.

To help maintain milk supply, a mother may pump 8 to 12 times per 24 hours, pump at the infant’s bedside, have the infant suckle on her emptied breast, or use kangaroo care. Maternal use of metoclopramide may increase milk production if a mother experiences a decline in her milk supply and the previously noted interventions do not help.

Most infants are discharged from the hospital when they are medically stable, taking all feedings orally, able to maintain their temperature, and are experiencing no apnea/bradycardia. Infants can be discharged on human milk or transitional or term formula. Depending on the NICU’s protocol, infants go home on either term formula or transitional formula. The calcium/phosphorous and caloric content of transitional formulas are between levels found in term and preterm formulas. Infants fed transitional formula for 9 months after NICU discharge have improved bone mineralization and growth. Breastfed and formula-fed infants drinking less than 500 mL in 24 hours need a multivitamin to provide 200 to 400 IU of vitamin D daily. Formula-fed
Shigella Species


Shigellae are gram-negative, aerobic bacilli that do not ferment lactose. Four species of Shigella cause gastrointestinal illness: S dysenteriae (group A including 13 serotypes), S flexneri (group B including 13 subserotypes), S boydii (group C including 18 serotypes), and S sonnei (group D comprising one serotype). Shigellae are transmitted readily person-to-person through fecal-oral and oral-anal contacts as well as indirectly through houseflies and contaminated fomites. Among the bacterial enteric pathogens, Shigella is unique because a small inoculum is capable of causing disease. Ingestion of as few as 10 organisms has caused illness in adult volunteers. The peak incidence of symptomatic Shigella infection occurs in 1- to 4-year-old children. Child care centers contribute to outbreaks through the grouping of susceptible children, a frequent lack of adherence to handwashing procedures, and the small inoculum required for disease production.

The typical incubation period is 2 to 4 days, with a range of 1 to 7 days. Shigella causes a range of gastrointestinal illness from mild diarrhea to life-threatening dysentery. Classically, children present with fever, severe abdominal cramps, and high-volume watery diarrhea (small bowel disease), followed 24 to 48 hours later by colitis with small-volume, bloody, mucoid stools and tenesmus (large bowel disease). Other children present with colitis, and, for unknown reasons, some children experience only a watery diarrhea that never progresses to colitis. Dysentery (or shigellosis) is more common with S dysenteriae and S flexneri infections; S boydii and S sonnei usually cause a watery diarrhea. Overall, only 40% of children who have Shigella infection have blood in their stools, and 50% have emesis. Seizures occur in 10% to 35% of hospitalized children who have shigellosis. This incidence is higher than would be expected from febrile seizures alone and suggests that shigellae produce a neurotoxin. In the past, it was believed that Shigatoxin caused these seizures, but it is now known that shigellae isolated from patients who have seizures do not produce Shigatoxin.

Physical findings may include a toxic-appearing child who has a high fever, lower quadrant abdominal tenderness, and tenderness on rectal examination. More than 66% of affected children have rectal temperatures higher than 102°F (38.9°C). Children who have shigellosis often are dehydrated and may have hyponatremia or hypoglycemia. Shigellosis causes a protein-losing enteropathy that can exacerbate malnutrition and lead to death. Rare complications of infection with Shigella include bacteremia, Reiter syndrome (after S flexneri infection), hemolytic-uremic syndrome (due to S dysenteriae type 1 infection), colonic perforation, and toxic encephalopathy.

When clinical findings suggest shigellosis, stool microscopy revealing large numbers of polymorphonuclear leukocytes supports the diagnosis. Definitive diagnosis is made with the isolation of Shigella sp from stool specimens or
rectal swabs. The stool specimen should be sent promptly to the laboratory because changes in stool pH occurring during prolonged transport may kill small numbers of shigellae. *Shigella* sp usually grow on MacConkey and Hektoen-Enteric agars. When low numbers of organisms are anticipated, a highly selective medium such as xylose-lysine-desoxycholate or *Shigella-Salmonella* medium is preferred. The positive culture rate in unselected stool specimens from patients who have diarrhea is about 2 per 100 specimens, with an approximate cost of $1,000 per positive stool culture. This cost decreases when laboratory testing is guided by clinical information and knowledge of the epidemiology of *Shigella* infection in a community.

Rehydration is the first step in treating affected children. Children who are not dehydrated and those who have been rehydrated should be fed an age-appropriate diet. Antimicrobial treatment is recommended for *Shigella* infections to shorten the course of the illness, decrease the duration of organism excretion, and decrease the secondary attack rate. Drug resistance is a serious problem with *Shigella*, making antimicrobial susceptibility testing of clinical isolates important. Quinolones are the preferred agents for treating *Shigella* in adults, but they are not approved for use in pediatric patients.

A third-generation cephalosporin is an option for empiric treatment of children until sensitivities are known. Parenteral ceftriaxone and oral cefixime have been used with success. Although some studies of cefixime in adults who had shigellosis were unimpressive, with only a 53% success rate, a study of a 5-day course of cefixime in Turkish children was more promising. An alternative is azithromycin, which has compared favorably with ciprofloxacin (82% versus 89% efficacy) in men who had shigellosis. Once antibiotic susceptibilities are known, therapy may be changed to ampicillin, trimethoprim-sulfamethoxazole, or nalidixic acid. For unclear reasons, in contrast to ampicillin, amoxicillin is not effective in treating *Shigella* infection.

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Comment: *Shigella* diarrhea is a worldwide problem that is associated with increasing antimicrobial resistance. Worldwide, the incidence of *Shigella* infection is highest in the toddler age group, although in the United States, *Shigella* accounts for fewer than 5% of diarrheal illness in this age group (Ramaswamy and Jacobson, 2001).

Because ingestion of only a few *Shigella* organisms can cause disease, transmission through a variety of mechanisms has been uncovered. Much transmission is through contaminated food and water that has included recent outbreaks from unchlorinated wading pools and recreational spray fountains. Person-to-person spread also occurs, especially in child care facilities and schools. Prevention includes handwashing; avoidance of contaminated food, water, and surfaces; and interruption of fecal-oral spread. One study using quantitative risk assessment found that adequate washing of hands after diapering reduces risk and can be reduced further by a factor of 20% by the use of an antibacterial soap (Gibson LL, et al. J Appl Microbiol. 2002;92(suppl):136S-143S).

Other preventive measures, especially in developing countries, include prolonged breastfeeding. It is postulated that breastfeeding may decrease shigellosis by preventing consumption of contaminated food and water, providing secretory immunoglobulin A and lactoferrin (thus decreasing bacterial virulence), increasing receptor glycolipids in the gut that bind Shigatoxin, or nonspecifically modifying the gut flora. Vaccines are in development.

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