Support Line

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Chair's Column

Ainsley Malone, MS, RD, CNSD

"In Pursuit of Excellence" — the theme of the recently concluded Food & Nutrition Conference and Expo (FNCE) in Philadelphia — couldn't better describe the activities and recognition experienced by DNS members who attended this annual professional event. The many outstanding educational events sponsored by DNS provided attendees with state-of-the-art information in nutrition support practice. The impressive presentations ranged from the Nestlé-sponsored workshop "Nutrition Screening – How Can We Improve Our Practice?" to the thought-provoking offering, "The Meaning of Food: Reflections from Home TPN-dependent Adults." What a great place to pursue excellence in the practice of nutrition support dietetics. DNS wishes to thank the following speakers for their contribution to this year's DNS offerings at FNCE: Pat Anthony, MS, RD; Pamela Charney, PhD, RD, CNSD; Johanna Dwyer, PhD, RD; Charlene Compher, PhD, RD, CNSD; Vanessa Kumpf, PharmD, BCNSP; Steve McClave, MD; David Thomas, MD; Naomi Trostler, PhD, RD; Vince Vanek, MD, CNSP; Susan Whitmire, RD, CNSD; and Marion Winkler, MS, RD, CNSD. Their commitment in both time and in providing high-quality education is greatly appreciated.

Excellence also was evident in the recognition of the extraordinary achievements of many DNS members at FNCE. These award recipients, recognized within this issue of *Support Line*, have and continue to represent the "crème de la creme" with regard to dietetics and nutrition support practice. DNS applauds each and every one of you. Thank you for what you have and will continue to contribute to our profession!

The pursuit of excellence continues with upcoming DNS educational events. In a few short weeks, the 2nd Annual DNS-Nestlé-sponsored Advanced Skills Workshop will be held in Pasadena, California, October 19-20, 2007. We look forward to another highly successful program and wish to thank Nestlé and the DNS Workshop Co-Chairs, Mary Marian, MS, RD, and Susan Roberts, MS, RD, CNSD, for their dedication and commitment to providing this outstanding educational event for our members. An abbreviated version of this workshop again will be held in conjunction with A.S.P.E.N.'s Clinical Nutrition Week in Chicago, February 9-13, 2008. In addition, as a participating organization in Clinical Nutrition Week, DNS is sponsoring several educational sessions. Watch your e-mailbox for further information. From educational offerings to products and publications, DNS leaders will continue to strive for excellence in all we do. In serving you, our members, we will offer nothing less.

Congratulation to DNS Members Honored at FNCE 2007

Medallion Recipients
Ronni Chernoff, PhD, RD, FADA, CSG
Marilyn Laskowski-Sachnoff, MA, RD

Excellence Awards
Clinical Practice: Susan Roberts, MS, RD, CNSD
Education: Gail P.A. Kauwell, PhD, RD
Research: Carrie P. Earthman, PhD, RD

DNS Distinguished Service Award
Charlene Compher, PhD, RD, CNSD

The Benefits and Safety of Probiotics in Infants

Theresa DeLorenzo, RD, CNSD

Abstract

Probiotics have multiple possible uses in infants and children. Various strains of these beneficial bacteria are available and have different safety profiles. Multiple studies document positive results in the use of probiotics to treat diarrhea in infants and necrotizing enterocolitis (NEC) in preterm infants, and they have the potential for improving growth rates. Probiotics are considered safe, but case reports of sepsis in immunocompromised infants suggest cautious use in this population. Further studies are needed to determine the most beneficial strains and the optimal dose.

Introduction

The term "probiotics" is derived from the Greek, meaning "for life" (1,2). The current definition is "living microorganisms, which upon ingestion in certain numbers, beneficially affect the host by improving its microbial balance and exerting health benefits beyond inherent general nutrition" (3). Probiotics can be referred to as functional foods because they provide health benefits beyond the traditional nutrition function (2). Most probiotics used today contain lactic acid-producing bacteria and typically belong to the *Lactobacillus*, *Bifidobacterium*, and *Streptococcus* groups (2,4,5). It is hypothesized that the beneficial effect of probiotics results from multiplication of the bacteria in the gastrointestinal tract, which subsequently displace pathogenic bacteria (6–9). Other proposed benefits include: 1) antagonism of pathogens by production of antimicrobial and antibacterial compounds such as cytokines, butyric acid, and other organic acids, thus reducing the number of pathogenic bacteria and inhibiting toxin production (7,9); 2) reduction in intestinal pH through the production of lactic acid (5,7,9); 3) competition for binding and receptor sites, thereby preventing colonization of pathogenic bacteria (7,9); 4) promotion of host defense by stimulating macrophage and natural killer cells; increasing secretory immunoglobulin (IgA) production and causing proliferation of lymphocytes (5,7,9); and 5) immune modulation by triggering cell-signaling events to stop virulence factors and sepsis (7–9). Probiotics also may aid lactose digestion through the production of lactase (9,10) and by delaying gastric emptying into the small intestine (2).

Applications

Sepsis

Probiotics have been used for the prevention and treatment of antibiotic-associated diarrhea and acute infantile diarrhea and may be effective for treatment of *Helicobacter pylori* infection (11). The use of probiotics is increasing in popularity as the incidence of sepsis rises. It is estimated that the rate of sepsis increases by 1.5% each decade and is the tenth most common cause of death in the United States (12). Preterm birth also is on the rise and is associated with a large proportion of the deaths related to NEC in the United States (12). Thus, preterm infants represent a target population for probiotic research.

Unlike breastfed term infants, preterm infants in intensive care units develop a very abnormal pattern of bowel colonization (13,14). Extensive use of antibiotics, infection control procedures, reduced exposure to maternal microflora, and sterile feedings all contribute to the development of abnormal colonization (15). The lack of microbial diversity in the bowels of preterm infants may predispose them to certain strains of sepsis such as those caused by staphylococci, Enterobacteriaceae, and enterococci (15). Probiotics can encourage bowel colonization with beneficial flora, thereby potentially protecting infants against life-threatening disease.

Necrotizing Enterocolitis

NEC is the most common abdominal emergency in preterm infants in intensive care units (15) and is potentially fatal. The abnormal pattern of gastrointestinal colonization of preterm infants may be a contributing factor to the pathogenesis of NEC (15). Changes in bacterial metabolic activities precede the onset of NEC, such as the fermentation of carbohydrates to produce intramural gas (16). The suggested benefits of probiotics are increased mucosal integrity, prevention of bacterial translocation, and improved tolerance to enteral nutrition, which can decrease the need for intravenous nutrition and regulation of the immune response, all of which contribute to reduced incidences of infection and intestinal disease (15).

Sakata and associates (17) found that the beneficial bifidobacteria commonly found in the term newborn gut were undetectable in the intestinal flora during the first 1 to 2 weeks after birth in preterm infants, even though they were being breastfed (18). The bacteria did not predominate until after the third postnatal week. Blakely and colleagues (13) documented similar findings, and Gewold and coworkers (14) observed bifidobacteria and lactobacilli in stools of fewer than 5% of extremely low-birth weight infants in the first week after birth. Caplan and colleagues (19) showed that bifidobacteria supplementation resulted in intestinal colonization and reduction in NEC-like lesions in a neonatal rat model. In a multicenter double-blind Italian study, preterm infants were randomized to receive a placebo or *Lactobacillus rhamnosus GG* (20). Researchers observed a decrease in NEC, although the difference was not statistically significant. A South American study showed a threefold decrease in NEC and a fourfold decrease in NEC-related mortality after the prophylactic administration of bifidobacteria-supplemented enteral

(Continued on next page)

feeding (21). In a prospective, randomized, blinded study, 367 breastfed preterm infants were randomized to *L acidophilus* and *B infantis* twice a day or no supplementation (22). A threefold decrease in NEC and NEC-related mortality followed prophylactic probiotic administration.

Diarrhea

Probiotics may prevent or ameliorate diarrhea through stimulation of the immune system, competition for binding sites on intestinal epithelial cells, and elaboration of bacteriocins (antibiotics produced by bacteria) (23). Infant rotavirus diarrhea can be treated effectively with *L rhamnosus GG* and *L casei Shirota* (24). *B bifidum* and *S therrmophilus* have successfully prevented rotavirus diarrhea in hospitalized infants (25). Gastroenteritis in infants has been treated effectively with *L rhamnosus GG* (26–30), *Enterococcus* SF68 (31), *L reuteri* (32), and *Saccharomyces boulardii* (33).

Antibiotic-associated diarrhea occurs in 11% to 62% of children who receive broad-spectrum antibiotics (34–36). Probiotics such as *Lactobacillus GG*, *S boulardi*, *E faecium*, *L acidophilus*, and *L bulgaricus* have been found to be effective for prevention and treatment (37–42). In a double-blind, placebo-controlled study involving infants and children with severe bacterial infections who received broad-spectrum antibiotics, the group that received probiotics had fewer episodes of diarrhea (37.5%) than the control group (80%) (43). In another study, researchers observed a significant reduction in hospital days and frequency of diarrhea as well as improved weight gain in children (6 to 24 months of age) who consumed yogurt compared with those who did not (435 g versus 383 g, P=0.017) (44).

Weight Gain

Additional studies (45–47) have investigated weight gain benefits of probiotics in infants. One randomized, controlled trial found that infants whose feedings were supplemented with *B breve* had higher rates of fecal bifidobacterial colonization at 2 weeks of age, decreased gastric aspirate volume, improved feeding tolerance, and enhanced weight gain (45). In a double-blind, randomized study, 120 healthy term infants up to 2 years of age received *L rhamnosus GG*-supplemented formula or regular formula and were followed until 6 months of age (46). The supplemented infants had significantly greater changes in length and weight and grew significantly better by the end of the study than the control group (P<0.01). The supplemented group also had notably more frequent defecation than the control group, suggesting that *L rhamnosus GG* may stimulate gut peristalsis and balance microflora and mucus production in a manner similar to that of breast milk. The greater frequency in stools did not result in diarrhea; the stools had formed consistency (46).

Availability

Probiotics are found in yogurt, specifically *Lactobacillus* and *Bifidobacterium*, which is the most common source for most of the population. To be beneficial to health, a probiotic must contain a "live" microbe (2). For infants and patients receiving nutrition support, capsule preparations are available (48). Probiotics also are available in powders and fermented milk products (1). Because probiotics do not adhere permanently to the intestine, daily supplementation appears to be necessary for effective therapy; the benefit disappears upon discontinuation of supplementation (9). Some products require refrigeration and often have shelf-lives of 3 to 6 weeks, but the concentration of viable bacteria may decrease with extended shelf-life (1,9).

Administration of at least 5×10^9 colony-forming units appears to be an effective probiotic dose (49), although the properties of different probiotic species vary, so the effects of one probiotic strain should not be generalized to others without confirmation in separate studies (2,50). Due to the lack of federal regulation, probiotic supplements produced by different manufacturers also differ widely (9). Hamilton-Miller and colleagues (51) analyzed the microbial content of 13 brands of probiotics in Britain and discovered that only two products matched their labeled microbial specifications. In a similar study of 15 probiotic products in the United States, researchers discovered that the number of viable cells identified in most of the products was significantly lower than reported on the label (52).

Safety

Although probiotics appear to be safe and have minimal adverse effects (9), caution should be exercised with certain patient groups, particularly preterm infants or patients who have immune deficiency or are immunosuppressed (6,50). Two case reports of children contracting serious infections have been documented in which the *L rhamnosus GG* strain isolated from the clinical specimen was indistinguishable from the probiotic strain administered to the patient (9,53,54). Poor perfusion due to inadequate cardiac function that compromised the gastrointestinal mucosa and facilitated the passage of the probiotic strain into the bloodstream has been proposed as the pathogenesis of infection (55). Another possibility is contamination of the central venous catheter during opening of the probiotic capsule or through hand-related transmission (55). Both patients improved when the infection was treated and the probiotic discontinued.

Conclusion

Probiotics such as *Lactobacillus GG* and *Bifidobacterium* have many proposed benefits in infants and children. Some of the benefits include prevention of sepsis, NEC, diarrhea, and infections and improved weight gain in premature infants. Although a variety of products are available, including capsules, powders, and foods, all products must contain "live" cultures to be effective. Caution should be exercised when administering probiotics to patients who are immunosuppressed due to case reports of children contracting serious illnesses after probiotic administration. Further study is necessary regarding the most optimal products and doses for the best outcomes for infants and children.

Theresa DeLorenzo, RD, CNSD, is a neonatal dietitian at Albany Medical Center, Albany, NY.

References

1. Cresci G. Probiotics: are they really "good little bugs?" *Support Line*. 2005; 27(1):20–27.
2. Lin D. Probiotics as functional foods. *Nutr Clin Pract.* 2003;18:497–506.
3. Bengmark S. Fiber (prebiotics) and probiotics: prevention of ICU infections with bioecological control and symbiotic treatment. In: Cresci G, ed. *Nutrition for the Critically Ill Patient: A Guide to Practice.* Boca Raton, Fla: CRC Press; 2005:151–172.
4. Gorbach SL. Probiotics and gastrointestinal health. *Am J Gastroenterol.* 2005;95(suppl):S8–S10.
5. Cresci GA. The use of probiotics with the treatment of diarrhea. *Nutr Clin Pract.* 2001;16:30–34.
6. Uterman V. Diet, nutrition and drug interactions In: Shils ME, Olson JA, Shike M, Ross CA, eds. *Modern Nutrition in Health and Disease*. 9th ed. Philadelphia, Pa: Lippincott Williams & Wilkins; 1999:1619–1641.
7. Bengmark S. Pre, pro and synbiotics. *Curr Opin Clin Nutr Metab Care*. 2001; 4:571–579.
8. Teitelbaum J, Walker W. Nutritional impact of pre- and probiotics as protective gastrointestinal organisms. *Ann Rev Nutr.* 2002;22:107–138.
9. Matarese LE, Seidner DL, Steiger E. The role of probiotics in gastrointestinal disease. *Nutr Clin Pract*. 2003;18: 507–516.
10. Madsen K. The use of probiotics in gastrointestinal disease. *Can J Gastroenterol.* 2001:15:817–822.
11. Bengmark S. Econutrition and health maintenance: a new concept to prevent GI inflammation, ulceration and sepsis. *Clin Nutr.* 1996;15:1–10.
12. Angus DC, Wax RS. Epidemiology of sepsis: an update. *Crit Care Med*. 2001: 29:109.
13. Blakey JL, Lutitz L, Barnes GL, et al. Development of gut colonization in preterm neonates. *J Med Microbiol.* 1982;15:519–529.
14. Gewold IH, Schwalbe RS, Taciak VL, et al. Stool microflora in extremely low birthweight infants. *Arch Dis Child Fetal Neonatal Ed.* 1999;80:F167–F173.
15. Millar M, Wilks M, Costeloe K. Probiotics for preterm infants. *Arch Dis Child Fetal Neonatal Ed.* 2003;88:F354.
16. Cheu HW, Brown D, Rowe MI. Breath hydrogen excretion as a screening test for the early diagnosis of necrotizing enterocolitis. *Am J Dis Child.* 1989;143:156–159.
17. Sakata H, Yoshioka H, Fujita K. Development of the intestinal flora in very low birth weight infants compared to normal full-term newborns. *Eur J Pediatr.* 1985;114:186–190.
18. Alona B, Bromiker R, Wilschanski M, et al. Oral probiotics prevent necrotizing enterocolitis in very low birth weight infants. *J Pediatr.* 2005; 54:192–196.
19. Caplan MS, Miller-Catchpole R, Caup S, et al. Bifidobacterial supplementation reduces incidence of enterocolitis in a neonatal rat model. *Gastroenterology.* 1999;117:577–583.
20. Dani C, Biadaioli R, Bertini G, Martelli E, Rubaltelli F. Probiotic feeding in prevention of urinary tract infection, bacterial sepsis and necrotizing enterocolitis in preterm infants. *Biol Neonate.* 2002;82:103–108.
21. Hoyos AB. Reduced incidence of necrotizing enterocoitis associated with enteral administration of *Lactobacillus acidophilus* and *Bifidobacterium infantis* to neonates in an intensive care unit. *Int J Inf Dis.* 1999;3:197–202.
22. Lin CH, Su BH, Chen AC, et al. Oral probiotics reduce the incidence and severity of necrotizing enterocolitis in very low birth weight infants. *Pediatrics.* 2005;115:171–172.
23. Jack RW, Tagg JR, Ray B. Bacteriocins of gram-positive bacteria. *Microbiol Rev.* 1995;59:171–200.
24. Sugita T. Togawa M. Efficacy of Lactobacillus preparation Biolactis powder in children with rotavirus enteritis. *Jpn Pediatr.* 1994;47: 2755–2762.
25. Saaedra JM, Bauman NA, Oung I., Perman JA, Yolken RH. Feeding of *Bifidobacterium bifidum* and *Streptococcus thermophilus* to infants in hospital for prevention of diarrhea and shedding of rotavirus. *Lancet.* 1994;344:1046–1049.
26. Guandalini S, Pensabene I, Zikri MA, et al. *Lactobacillus GG* administered in oral rehydration solution to children with acute diarrhea: a multicenter European Trial. *J Pediatr Gastroenterol Nutr.* 2000;30:54–60.
27. Raza S, Graham SM, Allen SJ, Sultana S, Cuevas L, Hart CA. *Lactobacillus GG* promotes recovery from acute non-bloody diarrhea in Pakistan. *Pediatr Infect Dis J.* 1995;14:107–111.
28. Pant AT, Graham SM, Allen SJ, et al. *Lactobacillus GG* and acute diarrhea in young children in the tropics. *J Trop Pediatr.* 1996;42:162–165.
29. Guarino A, Canani RB, Spagnuolo MI, Albano F, Di Benedetto L. Oral bacterial therapy reduces the duration of symptoms and vital excretion in children with a mild diarrhea. *J Pediatr Gastroenterol Nutr.* 1997;25:516–519.
30. Shornikova V, Isolauri E, Burkanoba L, Lukovnikova S, Vesikari T. *Lactobacillus GG* for treatment of acute diarrhea. *Acta Pediatr.* 1997;86:460–465.
31. Hellomo G, Mangiagle A, Nicastro L, Frigerio G. A controlled double blind study of SF68 strain as a new biological preparation for the treatment of diarrhea in pediatrics. *Curr Ther Res.* 1980;28: 927–936.
32. Shornikova V, Casas IA, Mykkanen H, Salo E, Veskari T. Bacteriotherapy with *Lactobacillus reteri* in rotavirus gastroenteritis. *Pediatr Infect Dis J.* 1997;16: 1103–1107.
33. Chapoy P. Treatment of acute diarrhea in children: a controlled trial with *Saccharomyces boulardii* [published in French]. *Ann Pediatrie.* 1985;32:1–3.
34. LaRosa M, Bottaro G, Gulino N, et al. Prevention of antibiotic-associated diarrhea with *Lactobacillus sporogenes* and fructo-oligosaccharides in children: a multi-centric double-blind vs. placebo study. *Minerva Pediatr.* 2003;55:447–452.
35. Turck D, Bernet JP, Marz J, et al. Incidence and risk factors for oral antibiotic-associated diarrhea in an outpatient pediatric population. *J Pediatr Gastroenterol Nutr.* 2003;37:22–26.
36. Estner CL, Lindsay AN, Book LS, et al. Lack of relationship of *Clostridium difficile* to antibiotic-associated diarrhea in children. *Pediatr Infect Dis.* 1983;2: 364–366.
37. Arvola T, Laiho K, Torkkeli S, Mykkanen H, Salminen S. Prophylactic *Lactobacillus GG* reduces antibiotic-associated diarrhea in children with respiratory infections: a randomized study. *Pediatrics.* 1999;104:64–68.
38. Vanderhoof J, Whitney D, Atonson D. *Lactobacillus GG* in the prevention of antibiotic-associated diarrhea in children. *J Pediatr.* 1999;135:564–568.
39. Surawicz C, Elmer G, Speelman P, McFarland L, Chin J, van Belle G. Prevention of antibiotic-associated diarrhea by *Saccharmyces boulardi*: a prospective study. *Gastroenterology.* 1989;96:981–988.
40. McFarland L, Surawicz C, Greenberg R, Elmer G, Moyer KA. Prevention of beta-lactam-associated diarrhea by *Saccaromyces boulardii* compared with placebo. *Am J Gastroenterol.* 1995;90: 439–448.
41. Wunderlich P, Braum L, Fumagalli I, D'Apuzzo B, Heim F. Double-blind report on the efficacy of lactic acid-producing *Enterococcus SF68* in the prevention of antibiotic-associated diarrhea and the treatment of acute diarrhea. *J Int Med Res.* 1989;17: 333–338.

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42. Gotz V, Romankiewicz J, Moss J, Murray H. Prophylaxis against ampicillin-associated diarrhea with a *Lactobacillus* preparation. *Am J Hosp Pharm.* 1979;36:754–757.
43. Jirapinyo P, Densupsoontorn N, Thamosiri N, Wongarn R. Prevention of antibiotic-associated diarrhea in infants by probiotics. *J Med Assoc Thai.* 2002;85(suppl 2)S739–S742.
44. Pashapour N, Iou SG. Evaluation of yogurt effect on acute diarrhea in 6-24 month old hospitalized infants. *Turk J Pediatr.* 2006;48:115–118.
45. Kitajima H, Sumida Y, Tanaka R, Yuki N, Takayama H, Fujimura M. Early administration of *Bifidobacterium breve* to preterm infants: a randomized controlled trial. *Arch Dis Child Fetal Neonatal Ed.* 1997;76:F101–F107.
46. Vendt N, Grunberg H, Tuure T, et al. Growth during the first 6 months of life in infants using formula enriched with *Lactobacillus rhamnosus GG*: double blind, randomized trial. *J Hum Nutr Diet.* 2006;19:51–58.
47. Berg RD. Probiotics, prebiotics or "conbiotics." *Trends Microbiol.* 1998; 6:89–92.
48. Murphy C, Vernon M, Cullen M. Intravenous immunoglobulin for resistant *Clostridium difficile* infection. *Age and Ageing.* 2006;35:85–86.
49. Johnston BC, Supina AL, Bohra S. Probiotics for pediatric antibiotic-associated diarrhea: a meta-analysis of randomized placebo controlled trials. *CMAJ.* 2006;175:1503–1516.
50. Boyle RJ, Robins-Browne RM, Tang ML. Probiotic use in clinical practice: what are the risks? *Am J Clin Nutr.* 2006;83:1256–1264.
51. Hamilton-Miller HM, Shah S, Winkler JT. Public health issues arising from microbiological and labeling quality of foods and supplements containing probiotic microorganisms. *Public Health Nutr.* 1999;2:223–229.
52. Canganella F, Paganini S, Ovidi M, et al. A microbiological investigation on probiotic pharmaceutical products used for human health. *Microbiol Res.* 1997; 16:43–51.
53. Rautio M, Jousimies-Somer H, Kauma H, et al. Liver abscess due to a *Lactobacilllus rhamnosus* strain indistinguishable from *L. rhamnosus* strain *GG*. *Clin Infect Dis.* 1999;28:1159–1160.
54. Ackay AD, Taylor MB, Kibbler CC, Hamilton-Miller JM. *Lactobacillus* endocarditis caused by a probiotic organism. *Clin Microbiol Infect.* 1999; 5:290–292.
55. Land MH, Rouster-Stevens K, Woods CH, Cannon ML, Cnota J, Shetty AK. *Lactobacillus* sepsis associated with probiotic therapy. *Pediatrics.* 2005; 115:178–181.

Intradialytic Parenteral Nutrition: A Nutrition Support Intervention for High-risk Malnutrition in Chronic Kidney Disease

Eileen Moore, RD, CNSD Stanley M. Lindenfeld, MD

Abstract

For the past decade, little evidence has emerged demonstrating a significant improvement in either morbidity or mortality of individuals receiving maintenance hemodialysis (MHD). It has been demonstrated clearly that severe malnutrition remains a significant problem for these patients and that hypoalbuminemia, reflected by serum albumin concentrations below 3.8 g/dL, is a strong predictor of both increased morbidity and mortality. Preventive strategies to improve the nutritional status of this group of patients are difficult to formulate due to the lack of definitive information regarding the biology of the dialytic state and its effect on factors that directly and indirectly control both appetite and important metabolic parameters related to appetite. Currently employed intervention strategies include diet liberalization, oral supplements, and enteral feeding, all of which often are ineffective in improving the nutritional status of the high-risk severely malnourished dialysis patient. For those patients who prove resistant to these measures and demonstrate either persistent severe protein malnutrition with albumin values below 3.5 g/dL or severe energy malnutrition with progressive weight loss, administration of intradialytic parenteral nutrition (IDPN) is re-emerging as a critically important clinical strategy. Evidence of its efficacy in treating severe malnutrition is increasing, and availability has expanded due to recent changes in coverage criteria. Notwithstanding the potential impact of IDPN, malnutrition in chronic kidney disease (CKD) stage 5 continues to pose a critical and challenging dilemma.

Introduction

Treatment of the patient who has stage 5 CKD and is receiving MHD has been improved both by technologic advances in dialysis treatment and advances in clinical management afforded by the National Kidney Foundation Kidney Disease Outcome Quality Initiative (K/DOQI) Guidelines (1). Panels of experts developed the comprehensive set of evidence-based guidelines that include respected expert opinion where evidence was insufficient. The purpose of the guidelines is to provide a framework for clinical decision-making. Guideline # 19 "Indications for Nutrition Support" is contained within the Clinical Practice Guidelines for Nutrition in Chronic Renal Failure. The guideline recommends consideration of dietary counseling, nutrition supplements, and tube feeding before parenteral nutrition in MHD patients.

Malnutrition in MHD: Uremia Influence

The incidence of malnutrition among patients receiving maintenance dialysis is believed to be between 18% and 70% (1). Dialysis patients often have poor appetites and low energy and dietary protein intakes (2). Factors contributing to the occurrence and/or persistence of malnutrition due to inadequate nutritional intake are summarized in the Figure. The dialysis procedure itself is catabolic, with losses between 5 and 8 g of amino acids per dialysis session and further losses (up to 10 g) when the dialyzer is reused (3). Uremia plays a prominent role in the development and/or persistence of malnutrition in CKD.

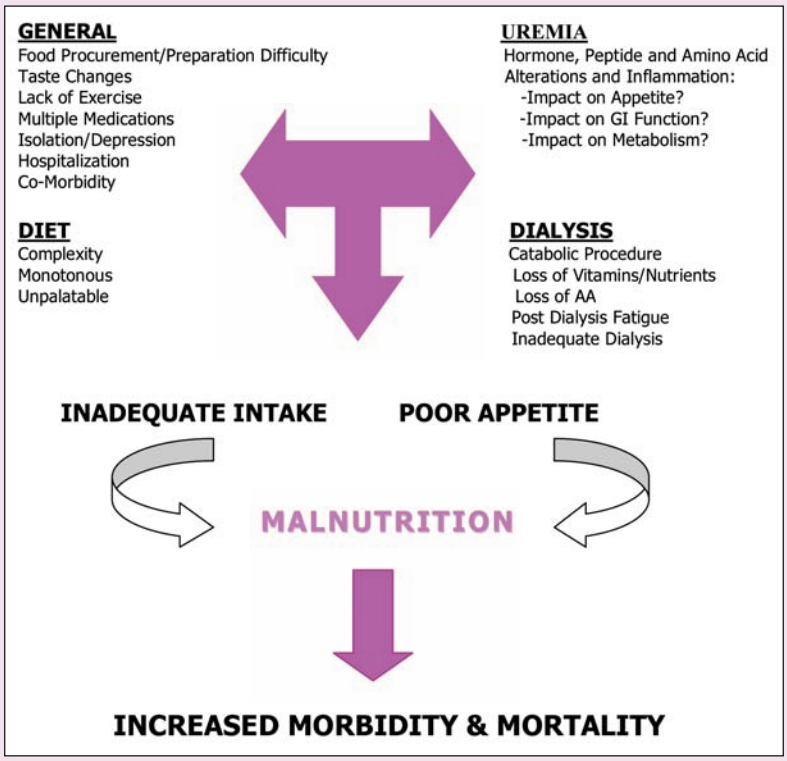
Uremia is associated with endocrine and metabolic alterations and inflammation that induce catabolic processes, inhibit anabolic processes, and influence regulatory processes on appetite. Metabolic alterations associated with uremia include chronic acidosis, which induces protein degradation and decreased synthesis of albumin (1). Components of uremia believed to play a role in the influence and/or regulation of appetite and gastrointestinal function include alterations in amino acid profile, hormones, and peptides and the presence of inflammation. The amino acid profiles in MHD patients are similar to amino acid profiles observed in non-CKD malnourished patients and include lower concentrations of branched-chain amino acids, which are believed to play a role in diminished appetite (4). Hormone and peptide elevations of leptin and cholecystokinin (CCK) may play a role in appetite regulation and nutrient intake. Leptin is speculated to contribute to decreased protein intake, loss of lean tissue and body weight, and an increase in energy expenditure. Inverse correlations between leptin and albumin values as well as protein intake have been demonstrated in those receiving MHD (5,6), although other investigations have not demonstrated correlations between leptin and lean body mass or nutrition parameters (7). Elevated CCK values have been demonstrated in patients receiving both MHD and peritoneal dialysis (PD) (8,9) and have been associated with satiety and decreased nutrient intake. Finally, inflammation is believed to play a role in appetite regulation. A chronic inflammatory state in MHD is characterized by elevated concentrations of tumor necrosis factor, interleukin-1 (IL-1), and IL-6. In cross-sectional studies, elevated C-reactive protein values were present in 22% to 53% of patients receiving MHD (10). In a recent study of 331 MHD patients, 38% subjectively reported "fair to poor" appetites (11). Anorexia was strongly associated with high levels of inflammatory markers and significantly associated with increased rates of hospitalization and higher mortality ($P<0.001$). More investigation is required to understand the complex nature of appetite regulation via orexigens and anorexigens that regulate the "satiety-hunger" cycle across the brain-gut axis.

Current Nutrition Interventions: Are They Working?

Evidence suggests that increases in protein intake (12) and albumin concentrations are associated with improved survival and that nutrition interventions designed to increase albumin concentrations to greater than 3.8 g/dL could reduce the number of deaths among those receiving MHD by approximately 10,000 per year (13). Currently, intensive counseling and use of the oral/enteral route of alimentation are employed for outpatient treatment of the malnourished MHD patient. A systematic review and meta-analysis of 18 studies of oral supplements and tube feedings included CKD patients receiving any form of maintenance dialysis in any setting and encompassed well-nourished and malnourished patients. Results showed that enteral nutrition increased total energy and protein intake and significantly increased albumin by 0.23 g/dL (14). The authors concluded that although data suggest that multinutrient support can increase total dietary intake significantly, correlation with improved clinical outcomes in malnourished (MHD) patients has not been established. Overall, minimal

(Continued on next page)

Figure. **Contributors to malnutrition in stage 5 chronic kidney disease.**



improvement in the attainment of desired serum albumin concentrations over a recent 10-year time period has been reported in the Clinical Performance Measures by the Medicare program (15).

The reason for the current ineffective standard approaches to nutrition interventions for MHD may have several components. First, there is lack of standardized intervention(s) and universally defined and accepted nutritional risk levels. Because the typical interventions are not specific to a risk level, differences in individual care plans may be ineffective due to lack of timely intervention or the underutilization of appropriate intervention(s). A recent survey of 196 MHD and PD outpatient practitioners sought to determine the prevalence of malnutrition using quantitative questions to identify indicators of malnutrition; examine interventions used to treat malnutrition; and share experiences, concerns, and solutions to problems in the management of malnutrition in dialysis patients (16). With respect to MHD patients, analysis of results showed: 17% of patients were considered malnourished; a significantly greater proportion of PD patients received oral supplements than MHD patients (P=0.0005); and there was a significant decrease in the use of tube feedings (P=0.000), IDPN (P=0.000), and PN (P=0.000) from previous to the current survey year.

A second factor is time commitment for intensive counseling and usual interventions (patient education, supplements, and coordinated care with nephrologists and social workers) that was demonstrated in a recent study (17). Authors indicated that due to reimbursement issues, current patient care ratios of approximately 100:1 are likely to increase, thereby further limiting time per patient. A third factor is barriers to oral supplement use and efficacy, which includes patient intolerance (16,18–20), lack of compliance (16,18), fluid restrictions (16,18), financial issues in procurement (10,16,18,20), and lack of encouragement from staff, including physicians (16).

Problems with enteral and parenteral alimentation routes represent a fourth factor. It appears that enteral feeding is underutilized in the dialysis population (16). Among the barriers to providing partial or total enteral tube feeding that have been identified are: lack of patient acceptance (16,19,20), intolerance (16,20), fluid constraints (16), patients who cannot be treated safely (medical or psychosocial disorders) (21), lack of assistance from family or caregivers (16), and financial issues (10,16). The barriers to PN encompass justified concern about using a potential future hemodialysis access site for nutrition (21), possibility of infection, and potential intolerance to fluid and minerals that may cause complications requiring additional dialysis treatments (21).

Of all the modalities available for nutrition support for the patient receiving MHD, IDPN is an efficient, convenient, assured route of supplemental alimentation. The modality allows for ultrafiltration during dialysis for fluid management, no reliance on patient adherence, and no need for a dedicated feeding tube or vascular access (1,18,20,21,22). However, the therapy is not without controversy.

IDPN Therapy: History and Recent Evidence of Efficacy

IDPN is infused during the hemodialysis procedure through the tubing of the venous drip chamber. IDPN has been used for several decades, with early investigation directed toward the correction of an abnormal amino acid profile associated with MHD in an attempt to improve appetite (23). Weight gain and improved aminograms resulted from the use of general amino acid solutions, although the use of essential amino acids resulted in a deficit of nonessential amino acids, which was believed to be unfavorable for protein anabolism (24,25).

A recent evidence-based review of IDPN noted numerous flaws in available studies that limited their usefulness, but concluded that IDPN therapy is reasonable for patients selected by published criteria of severe malnutrition (26). Further, two studies and one retrospective analysis (rated as level C data) suggested that IDPN therapy is associated with decreased mortality.

Recent studies using labeled isotopes (which allows for assessment of protein turnover and energy balance) provide evidence of significant positive effects of IDPN on protein and energy metabolism (22). The results of infusion of leucine and phenylalanine before during and after hemodialysis treatment of seven well-nourished patients studied for one session with IDPN versus one session without IDPN showed that provision of calories and amino acid during hemodialysis with IDPN reversed a net negative whole body and forearm muscle protein balance. Essentially, IDPN compensated for the catabolic effects of the dialysis procedure by stimulating muscle protein synthesis and promoting a positive net skeletal and muscle protein balance. None of these beneficial effects carried over to the postdialysis period; both protein synthesis and proteolysis returned toward baseline levels shortly after IDPN was discontinued. The same isotope techniques applied in a randomized, crossover study on seven well-nourished MHD patients receiving IDPN resulted in an improved fractional synthetic rate of albumin during MHD, which paralleled improvements in whole-body protein synthesis (27). Despite the small sample size in these two studies, they provide intriguing information on the detailed metabolic consequences of IDPN (protein anabolic), which could provide insight into the influence of IDPN on outcome if studied over a longer period of time.

Reimbursement History

Based on experience with PN in the hospital setting, attempts to supplement the nutritional intake of dialysis patients by providing nutrients during the dialysis procedure were found to be beneficial in studies done in the late 1980s and early 1990s (25,28–32). Coverage initially was provided as a subtype of Parenteral and Enteral Nutrition (PEN) administered by two specialty PEN carriers that were reimbursement agencies of Medicare. In 1993, the Health Care Financing Administration (HCFA) created four DMERCs (disposable medical equipment

regional carriers) with new guidelines, making the criteria identical to that of PEN. Effective October 1, 1993, the coverage policy for PEN/IDPN was rewritten, making the coverage criteria identical to that of total parenteral nutrition. The criteria required the following conditions to be present: 1) permanent malfunction of the gastrointestinal tract, resulting in significant malabsorption; 2) malnutrition such that therapy was required to sustain life; and 3) unavailability of a practical, less extreme intervention, such as oral supplements or enteral nutrition. All patients who had been on service before the 1993 policy took effect were "grandfathered in" to avoid any disruption in care (28).

Physician prescribers broadly interpreted the requirements, as reflected in a significant variation of the percentage of patients who were placed on therapy among different providers (28). IDPN became an established therapy to replenish malnourished MHD patients in the early 1990s, with utilization figures varying from 0 to 25% of the dialysis population (28). Initial clinical experience was positive, with some studies suggesting a potential to improve morbidity and mortality in this population (31,33). However, concerns over the cost of therapy and the lack of any large, well-designed prospective studies to prove the impact of IDPN led Medicare to restrict the availability of these therapies severely in July 1996 and to discontinue their availability to patients previously receiving IDPN who no longer met the newer restricted guidelines (28).

In the late 1990s, Medicare Part B covered only IDPN therapy in patients who had clearly documented permanent impairment of the gastrointestinal tract accompanied by proven malabsorption of vital nutrients (28). This statutory restriction essentially limited access to IDPN to an extremely small group of patients.

With recognition of the scope and magnitude of malnutrition accompanying MHD and the increasing evidence of the positive impact of IDPN (26,34,35), a more positive reimbursement environment has emerged. In 2005, a consortium of commercial health plans revisited the rationale for IDPN and broadened the criteria to include patients unable to meet the protein requirements established for MHD patients through oral intake and oral protein supplements (36,37). With the implementation of Medicare Part D in 2006, Part D Plans were instructed to cover their beneficiaries for IDPN when the patient did not qualify under the restricted Part B guidelines through an individual exception process if there was clinical documentation of severe malnutrition not amenable to oral intake of foods (Personal communication, Dr. Jeffrey Kelman, CMS Chief Medical Officer for the Center for Beneficiary Choices, March, 2006). As a result, the use of IDPN has increased steadily. In January 2007, amino acid solutions for IDPN were added to the Medicare Part D formulary, and patients now are approved through a prior authorization process rather than an exception process. To clarify the appropriateness of Part D coverage further, Centers for Medicare & Medicaid Services formally changed a section of the Part D manual in early 2007, clearly stating specifically that IDPN was covered under Part D when the patient did not qualify under the Part B requirements (38).

Current Coverage Criteria for IDPN

Current criteria for coverage for IDPN vary somewhat, depending on the commercial health insurance provider or the individual Part D provider. However, the criteria are sufficiently similar to identify general guidelines. First, the criteria for coverage under Medicare Part B (i.e., permanent impairment of the gastrointestinal tract leading to malabsorption as the primary cause of malnutrition) now apply only to coverage under Part B; they have no role in the coverage determination for Part D or commercial coverage (38). Patients who have severe malnutrition, as indicated by clinical evidence discussed later in this article, now are eligible for coverage. To receive coverage for "protein" malnutrition, patients generally must have documented persistent albumin concentrations less than 3.5 g/dL and protein intakes less than 0.8 g/kg body weight and have failed to respond to dietary counseling or attempts at oral supplementation (36,37). To qualify for energy malnutrition, patients generally must have evidence of significant recent weight loss (i.e., >5%, body mass index <18, or weight less than 90% of the ideal body weight) and have not responded to dietary counseling or caloric supplementation.

It is now recognized that patients receiving MHD may have evidence only of protein malnutrition without energy malnutrition (1,36). Such patients may consume adequate calories to maintain weight, but due to increased protein requirements enforced by dialysis, they may not be able to ingest quantities of protein close to that recommended by K/DOQI. When this situation leads to clinical evidence of severe protein malnutrition, IDPN is considered appropriate therapy by a growing number of nephrologists and is covered by many commercial plans and Medicare Part D (36–38, personal communication, Dr. Jeffrey Kelman, March 2006).

Current Clinical Practice of IDPN

Identifying the IDPN Patient

In addition to the coverage criteria discussed previously, K/DOQI Nutrition Guidelines outline three criteria to determine the malnourished MHD patient who may benefit from IDPN: 1) evidence of malnutrition and inadequate dietary intake, 2) evidence of the inability to administer or tolerate adequate oral nutrition inclusive of supplements and tube feeding, and 3) the need to combine oral or enteral intake with IDPN to meet the individual's nutrition needs (1). The National Kidney Foundation guidelines position paper on proposed HCFA guidelines for reimbursement of enteral nutrition and PN references information on patient selection and nutrition intervention with progression to parenteral alimentation routes (21). IDPN clearly is not a first line of nutrition intervention. Caloric provision via IDPN cannot provide for *total daily* estimated requirements and, therefore, is not a

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substitute for PN when individuals require daily nutrition needs to be met. Further, patients with severe medical problems that contribute to significant weight loss and/or protein depletion (e.g., cancer, nephrotic proteinuria) need individual physician evaluation as to the appropriateness of IDPN therapy.

Design of IDPN Therapy

Optimal IDPN solution composition has not been defined, and published "generally accepted" solutions (39,40) may not always be good selections for patients who are very malnourished or have low body weights (41,42). Safe design for IDPN therapy has its basis in PN literature. Individualized substrate utilization rates should be considered; understanding of the impact of substrates is imperative for safe and effective IDPN provision. The Table provides examples of "generally accepted" solutions and tailored solutions.

Glucose: Glucose clearance and oxidation vary, with maximal clearance ranging from 3 to 9 mg/kg/min (43). The typical IDPN solution glucose component for patients requiring carbohydrate control is 4 to 6 mg/kg/min and 6 to 8 mg/kg/min for those not requiring carbohydrate control. The potential for insulin resistance, hyperglycemia, or hypoglycemia exists, suggesting the need for appropriate monitoring of blood glucose concentrations. Sliding scale coverage with regular insulin may be employed for the first or "mid-run" (1 hour into infusion) arterial glucose concentrations. Insulin may be administered subcutaneously or added to the IDPN solutions prior to or during administration, as directed by physician (44,45). The patient's current glucose control regimen should be considered. Hypoglycemia can be prevented by consumption of a snack during the last 30 minutes of treatment (44,45). Patients who have received insulin during IDPN should be monitored closely.

Amino Acids: Amino acids usually are provided at 1.2 to 1.4+ g/kg protein using general amino acids (33). Patients who have a history of protein intolerance require decreased amounts of protein provided as tolerated. Amino acid provision has the potential for acidosis, whose deleterious effects were described previously. Monitoring of serum bicarbonate or carbon dioxide concentrations is recommended, with oral bicarbonate or acetate salt added to IDPN at the physician's discretion. Amino acids provision may result in an increased urea volume, which may influence Kt/V (46). Kt/V measures the fractional clearance of urea as a function of its volume distribution in the body and is used to assess dialysis adequacy. Whether this impact is valid in terms of assessing dialysis adequacy remains to be clarified. If problematic, Kt/V may be measured during a dialysis session where IDPN is withheld to assess the clearance more accurately and the need for a change in dialysis prescription.

Intravenous (IV) Lipids: An abnormal lipid profile is associated with CKD. In a study of 240 patients who had CKD, blood analysis of lipid parameters after an overnight fast revealed significantly higher triglyceride (TG) and lower high-density lipoprotein and Apo-1 concentrations compared with controls (47). Serum carnitine deficiency, elevated concentrations of free fatty acids (FFAs), and disordered fatty acid metabolism also are seen in MHD patients (48). Provision of carnitine to MHD patients has been shown to decrease plasma FFAs, and although not all studies have demonstrated decreases in serum TGs, investigation and correction of carnitine deficiency for patients with elevated TG concentrations may be prudent.

Lipid abnormalities may raise concerns about the use of IV lipid in IDPN regimens. An IV fat tolerance test administered to 17 controls, 25 patients who had chronic renal insufficiency (CRI), and 32 patients receiving MHD showed decreased fractional removal rate of IV lipid in the CRI and MHD patients that was inversely related to serum TG and fasting blood glucose concentrations compared with controls (49). Another concern with IV lipid provision is the potential for FFA toxicity. FFA concentrations are elevated in dialysis patients. Because FFAs are bound to albumin, patients who have low albumin concentrations may have elevated concentrations of FFAs. FFA toxicity can cause cardiac dysfunction, arrhythmias, and pulmonary injury (42). Although this has not been specifically reported in IDPN literature, it is unknown what level of albumin has the potential to elicit this reaction (personal communication, Dr. Brian Schreiber, August 2007).

Judicious provision of IV lipid in IDPN is usually the lower of either 4 mg/kg/min or 12 to 12.5 g/hr, with observation for tolerance (50). The initial lipid-containing IDPN solution should be infused at 1 mL/min for 10% IV lipid or 0.5 mL/min for 20% IV lipid for 30 minutes with assessment of tolerance (44,45) before advancing to the prescribed goal rate.

Fluids: IDPN generally is infused in less than a 1-L volume. Astute clinical and physical assessment by the dialysis nurse can guide fluid removal for the dialysis session. As IDPN therapy continues and true weight gain results, fluid removal decisions and assessment of estimated dry weight are necessary.

Micronutrients: Addition of micronutrients is not standard practice, although the addition of vitamins and minerals should be considered for patients who do not tolerate oral vitamin supplementation and who exhibit higher degrees of malnutrition because they may have depleted reserves. Abnormal renal metabolism, inadequate intake, or gastrointestinal and dialysis losses contribute to vitamin and mineral deficiencies in MHD patients. As with those receiving long-term PN, it can be a challenge to assess for micronutrient deficiency in malnourished dialysis patients (51). Water-soluble vitamins should be supplemented daily. Vitamin and mineral disturbances in dialysis patients most often involve vitamin C, folic acid, vitamin B_1 and B_6, zinc, selenium, and recently manganese (52–56). Clinical manifestations of such disturbances include depressed immune system, neuropathy, impaired amino acid and lipid metabolism, mild scurvy, and other abnormalities. Clinical manifestations of vitamin, mineral, and

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Table. **Sample IDPN Solutions and Infusion Schedules**

Sample "STANDARD SOLUTIONS"*

Characteristic	Prescription	Total Volume	Formula Composition	Total Kcal
Prescription 1	250 mL D50 250 mL 10% AA	500 mL	125 g dextrose 25 g protein	525
Prescription 2	250 mL D50 550 mL 10% AA 250 mL 20% Lipid	1,050 mL	125 g dextrose 55 g protein 50 g fat	1,145
Rate of Infusion: Divide IDPN volume by dialysis treatment time. Do not exceed 350 mL/hr.				
Prescription 2b Lipid-free	500 mL D50 550 mL 10% AA	1,050 mL	250 g dextrose 55 g protein	1,070
Rate of Infusion: Divide IDPN volume by dialysis treatment time. Do not exceed 350 mL/hr.				
Prescription 2c Volume control	250 mL D70 550 mL 10% AA	800 mL	175 g dextrose 55 g protein	815
Rate of Infusion: Divide IDPN volume by dialysis treatment time. Do not exceed 350 mL/hr.				

*In general: Prescription 1 infused for three treatments or until tolerated, then Prescription 2. Prescription 2b is for lipid-intolerant patients. Prescription 2c is for patients requiring volume control.

Adapted from Goldstein J. Intradialytic parenteral nutrition evolution and current concepts. *JRN.* 1991;1(9).

Sample "TAILORED SOLUTIONS"

Sample IDPN based on EDW: 60 Kg 3.5 hr hemodialysis (HD) treatment

6 mg/kg carbohydrate (CHO) control (diabetic) 8 mg/kg standard CHO provision and 1.4+ g/kg protein

Characteristic	Prescription	Total Volume	Formula Composition	Total Kcal
High-protein CHO control Lipid-free	108 mL D70 *560 mL 15% AA	703 mL (with 35 mL fill)	75.6 g dextrose 84 g protein	593
Rate of Infusion: Divide half-volume IDPN by HD treatment time for three treatments (~100 mL/hr) If tolerated, increase to full volume. Divide full-volume IDPN by HD treatment time (200 mL/hr).				
High-protein Standard CHO Lipid-free	144 mL D70 *560 mL 15% AA	739 mL (with 35 mL fill)	100.8 g dextrose 84 g protein	679
Rate of Infusion: Divide half-volume IDPN by HD treatment time for three treatments (~110 mL/hr) If tolerated, increase to full volume. Divide full-volume IDPN by HD treatment time (215 mL/hr).				
High-protein High-calorie	144 mL D70 *560 mL 15% AA **210 mL 20% lipid	949 mL (with 35 mL fill)	100.8 g dextrose 84 g protein 42 g fat	1,099
Rate of Infusion: Divide half-volume CHO & AA IDPN by HD treatment time for three treatments (~110 mL/hr). If tolerated, increase to full-volume CHO and AA. Divide volume by HD treatment time (215 mL/hr). *** *Test dose first IDPN lipid-containing bag for 30 min (135 mL/hr).* If tolerated, increase to full-volume CHO, AA, lipid. Divide volume by HD treatment time (275 mL/hr).				

Solution selection based on: need for CHO control, goals of therapy (protein repletion versus protein and calorie repletion), and lipid tolerance.

Vitamins (without aluminum), trace elements, minerals, electrolytes may be prescribed (see text).

*May use higher base of 20% AA for further fluid conservation.

**May use higher base of 30% lipid for further fluid conservation.

nutrient deficiency states as well as increased anabolic needs should be addressed individually. When supplementing micronutrients in IDPN, monitoring for deficiency and toxicity should be similar to that for PN (51). Potential for aluminum excess and toxicity in renal patients warrants prudent PN vitamin selection with proper monitoring of plasma aluminum concentrations (57). Due to the nature of loss through hemodilaysis, vitamins may be added to the IDPN bag during the last 30 minutes of infusion. Trace elements can be added by the pharmacy.

Administration and Monitoring

Effective administration and monitoring of IDPN in the dialysis unit requires additional time and effort for physician, nurse, and dietitian. Unit clinicians should be educated via in-services that may include infusion pump procedures, storage and handling procedures, and administration of IDPN, including procedures with vitamins and medications. Provider guidelines are general, and clinicians should request pharmacist assistance for individual considerations.

IDPN solutions should be initiated at low rates of infusion, particularly for patients at risk of refeeding syndrome (58). Physical signs of a suboptimal dialysis treatment, including nausea, vomiting, or hypotension, should be evaluated carefully against the patient's "usual" treatments. Symptoms may be due to the cold temperature of the administered solution, volume or substrate intolerance, electrolyte imbalance, or nutrient deficiencies (34,39,44,45). Prudent laboratory monitoring and patient observation allows appropriate identification of the cause, allowing corrective action. When indicated, sodium, phosphorus, potassium, and magnesium may be added by changing the dialysate bath or by adding oral medications or salts to IDPN as directed by the physician.

Physical and nutrition assessment with close monitoring of biochemical laboratory parameters and dry weight are integral to providing effective IDPN. Although patient-specific, nutritional gain from IDPN typically requires 3 to 6 months (28,35). No data are available regarding either the longevity of the beneficial impact of IDPN or consequent nutritional status of patients in whom IDPN is discontinued.

Future of IDPN Therapy

The treatment of malnutrition among the dialysis population has been a significant challenge. Significant questions and controversy regarding both the clinical impact of this problem and the most effective approach to it remain. Nonetheless, it is possible to affect this critical condition. The ability to provide IDPN and have it reimbursed either through Medicare Part D or commercial payers should change substantially the use and understanding of the role of this potentially important therapy.

IDPN specifically answers the needs of patients who cannot meet their requirement of nutrient intake through currently available means. Greater experience with this therapy combined with ongoing monitoring of the clinical response, the ability to provide therapy specifically tailored to individual patient needs, and the use of appropriate substrate utilization principles should maximize attainment of the desired nutritional outcomes. Further study should increase the ability to define critical determinants of responsiveness to IDPN, and carefully designed prospective studies should define further the science of nutritional repletion. Attempts to identify and understand the barriers preventing adequate dietary intake of nutrients, where appropriate, for this population will continue. Future research in the area of naturally occurring appetite stimulants and suppressants and their relationship to the nutritional state of the dialysis population eventually may lead to the ability to prevent or more effectively treat the development of severe malnutrition. Until this knowledge is available and applicable, the increased availability and maximization of IDPN therapy is a significant and positive step toward appropriate and effective care of this at-risk population. Currently, an interorganizational effort is underway to develop "Expert Practice Recommendations for Intradialytic Parenteral Nutrition (IDPN) and Intraperitoneal Nutrition (IPN)." A group of individuals representing recognized renal and nutrition support organizations with therapy expertise will develop recommendations for practitioners to identify high-risk hemodialysis and PD patients appropriate for these therapies and provide much needed therapy practice recommendations.

Conclusion

Defining and effectively addressing factors attributed to malnutrition in MHD remains a perplexing and challenging dilemma. The lack of improvement in the nutritional status of MHD patients over the past decade suggests the need for a new direction. Integral to this direction is the development and study of designed/tiered nutrition interventions, including IDPN, where the intervention(s) is commensurate with identified problems and malnutrition/risk levels. Of equal importance to IDPN therapy is enhancement of clinician nutrition support knowledge and skill to apply effective nutrition support therapy (enteral nutrition, IDPN, and PN). Certainly, the effort underway for "Expert Practice Recommendations for IDPN and IPN" will be an important step toward achieving these objectives in the attempt to improve outcomes of dialysis patients.

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References

1. National Kidney Foundation. K/DOQI clinical practice guidelines for nutrition in chronic renal failure. *Am J Kidney Dis.* 2000;35(suppl 2):S1–S140.
2. Rocco MV, Paranandi L, Burrowes JD, et al. Nutritional status in the HEMO Study cohort at baseline hemodialysis. *Am J Kidney Dis.* 2002;39:245–256.
3 Ikizler TA, Flakoll P, Parker R, Hakim R. Amino acid and albumin losses during hemodialysis. *Kidney Int.* 1994;46: 830–837

4. Skeie B, Manner T, Lynn R, Ginsberg N, Katz DP, Askanazi J. Intradialytic parenteral nutrition. *Dial Transplant.* 1991;20:551–552.
5. Dascher M, Tonshoff B, Blum WF, et al. Inappropriate elevation of serum leptin levels in children with chronic renal failure. *J Am Soc Nephrol.* 1998; 9:1074–1079.
6. Johansen KL, Mulligan K, Tai V, Schambelan M. Leptin body composition, and indices of malnutrition in patients on dialysis. *J Am Soc Nephrol.* 1998;9:1080–1084.
7. Yilmaz A, Kayardi M, Icagasioglu S, Canden F, Nur N, Gultekin F. Relationship between serum leptin levels and body composition and markers of malnutrition in nondiabetic patients on peritoneal dialysis or hemodialysis *J Chi Med Assoc.* 2005;68:566–570.
8. Aguilera A, Codoceo R, Selgas R, Garcia P, et al. Anorexigen (TNF-α), cholecystokinin and oregxigen (neuropeptide Y) plasma levels in peritoneal dialysis (PD) patients: their relationship with nutritional parameters. *Nephrol Dial Transplant.* 1998;13:1476–1483.
9. Wright M, Woodrow G, O'Brien S, et al. Cholecystokinin and leptin: their influence upon the eating behaviour and nutrient intake of dialysis patients. *Nephrol Dial Transplant.* 2004;19:133–140.
10. Mehrotra R, Kopple J. Nutritional management of maintenance dialysis patients: why aren't we doing better? *Annu Rev Nutr.* 2001;21:343–380.
11. Kalantar-Zadeh K, Block G, McAllister C, Humphreys M, Kopple J. Appetite and inflammation, nutrition, anemia, and clinical outcome in hemodialysis patients. *Am J Clin Nutr.* 2004;80: 299–307.
12. Shinaberger C, Kilpatrick R, Regidor D, et al. Longitudinal associations between dietary protein intake and survival in hemodialysis patients. *Am J Kidney Dis.* 2006;49:37–49.
13. Kalantar-Zadeh K, Kilpatrick R, Kuwae N, et al. Revisiting mortality predictability of serum albumin in the dialysis population: time dependency, longitudinal changes and population-attributable fraction. *Nephrol Dial Transplant.* 2005;20:1880–1888.
14. Stratton R, Bircher G, Fouque D, et al. Multinutrient oral supplements and tube feeding in maintenance dialysis: a systematic review and meta-analysis. *Am J Kidney Dis.* 2005;46:387–405.
15. Centers for Medicare and Medicaid Services. *2004 Annual Report, ESRD Clinical Measures Performance Project.* Baltimore, Md: Department of Health and Human Services, Centers for Medicare and Medicaid Services; 2004.
16. Wai Chi Tai T, Chan A, Cochran C, et al. Use of intradialytic parenteral nutrition: an end stage renal disease network perspective. *J Ren Nutr.* 1994;4:11–14.
17. Leon J, Albert J, Gilchrist G, et al. Improving albumin levels among hemodialysis patients: a community-based randomized controlled trial. *Am J Kidney Dis.* 2006;48:28–36.
18. Hiroshige K, Iwamoto M, Kabahima N, Mutoh Y, Yuu K, Ohtani A. Prolonged use of intradialysis parenteral nutrition in elderly malnourished chronic hemodialysis patients. *Nephrol Dial Transplant.* 1998;13:2081–2087.
19. Chan A, Cochran C, Harbert PT, Foulks C, Lindley J. Use of intradialytic parenteral nutrition: an end stage renal disease network perspective. *J Ren Nutr.* 1994;4:11–14.
20. Hakim R, Levin N. Malnutrition in hemodialysis patients. *Am J Kidney Dis.* 1993;21:125–137.
21. Kopple J, Foulks C, Piraino B, Beto J, Goldstein J. Proposed Health Care Financing Administration guidelines for reimbursement for enteral and parenteral nutrition. *Am J Kid Dis.* 1995;26:995–997.
22. Pupim L, Flakoll P, Brouillette J, Levelhagen D, Hakim R, Ikizler A. Intradialytic parenteral nutrition improves protein and energy homeostasis in chronic hemodialysis patients. *J Clin Invest.* 2002;110:483–492.
23. Powers D. Prolonged experience with intradialytic hyperalimentation in marasmic chronic hemodialysis patients. *Contemp Dial Nephrol.* 1989;May:22–28.
24. Piraino A, Firpo J, Powers D. Prolonged hyperalimentation in catabolic chronic dialysis therapy patients. *JPEN J Parenter Enteral Nutr.* 1981;5:463–477.
25. Powers D, Jackson A, Piraino A. Prolonged intradialysis hyperalimentation in chronic hemodialysis patients with an amino acid solution (RenAmin Amino Acid Injection) formulated for renal failure. In: Kinney J, Borum P, eds. *Perspectives in Clinical Nutrition.* Baltimore, Md: Urban & Schwarzenberg; 1989:191–205.
26. Foulks CJ. An evidence-based evaluation of intradialytic parenteral nutrition. *Am J Kidney Dis.* 1999;33:186–192.
27. Pupim L, Flakoll P, Ikizler TA. Nutritional supplementation acutely increases albumin fractional synthetic rate in chronic hemodialysis patients. *J Am Soc Nephrol.* 2004;15:1920–1926
28. Lazarus, MJ. Recommended criteria for initiating and discontinuing intradialytic parenteral nutrition therapy. *Am J Kidney Dis.* 1999;33:211–216.
29. Bilbrey GL, Cohen T. Identification and treatment of protein calorie malnutrition in chronic hemodialysis patients. *Dial Transplant.* 1989;8:669–677.
30. Foulks CJ. The effect of intradialytic parenteral nutrition on hospitalization rate and mortality in malnourished hemodialysis patients. *J Renal Nutr.* 1994;4:5–10.
31. Capelli JP, Kushner H, Camiscioli TC, Chen SM, Torres MA. Effect of intradialytic parenteral nutrition on mortality rates in end-stage renal disease care. *Am J Kidney Dis.* 1994;23:808–816.
32. Snyder S, Bergen C, Sigler MH, Teehan BP. Intradialytic parenteral nutrition in chronic hemodialysis patients. *ASAIO Trans.* 1991;37:373–375.
33. Chertow GM, Ling J, Lew NL, Lazarus JM, Lowrie EG. The association of intradialytic parenteral nutrition administration with survival in hemodialysis patients. *Am J Kidney Dis.* 1994;24:912–920.
34. Hiroshige K, Iwamoto M, Kabashima N, Mutoh Y, Yuu K, Ohtani A. Prolonged use of intradialysis parenteral nutrition in elderly malnourished chronic haemodialysis patients. *Nephrol Dial Transplant.* 1998;13:2081–2087.
35. Cherry N, Shalansky K. Efficacy of intradialytic parenteral nutrition in malnourished hemodialysis patients. *Am J Health-Syst Pharm.* 2002;59: 1736–1741.
36. *Intradialytic Parenteral Nutrition and Intraperitoneal Nutrition, Blue Cross of California.* Available at: http://medpolicy.bluecrossca.com/policies/MED/intradialytic_parenteral_nutrition.html.
37. *CIGNA Healthcare Coverage Position.* Available at: http://www.cigna.com/customer_care/healthcare_professional/coverage_positions/medical /mm_0136_coveragepositioncriteria_nutritional_support.pdf.
38. Chapter 6, (Appendix C-1). *Medicare Part D Manual.* 2007;Oct:8.
39. Goldstein D, Strom J. Intradialytic parenteral nutrition: evolution and current concepts. *J Renal Nutr.* 1991; 1:9–22.
40. McCann L. Malnutrition in chronic kidney disease. In: *Pocket Guide to Nutrition Assessment of the Patient with Chronic Kidney Disease.* 3rd ed. New York, NY: National Kidney Foundation; 2002:15–21
41. Russell GI, Davies TG, Walls J. Evaluation of the intravenous fat tolerance test in chronic renal disease. *Clin Nephrol.* 1980;13:282–286.
42. Powers D. Considerations in the use of 3:1 intradialytic parenteral nutrition solutions containing long-chain triglyceride. *Cont Dial & Nephrol.* 1990; February:29–34.
43. Wolfe R. Carbohydrate metabolism and requirements. In: Rombeau JL, Caldwell MD, eds. *Parenteral Nutrition: Volume 2.* Philadelphia, Pa: WB Saunders; 1986:53–71.

(Continued on page 16)

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44. *Comprehensive Renal Nutritional Support Program Clinical Manual of FMC.*
45. *What Are the Recommended Patient Monitoring Protocols? In IDPN Services.* Deerfield Ill: Baxter Renal Division, IDPN Services.
46. McCann L, Feldman C, Hornberger J, et al. Effect of intradialytic parenteral nutrition on delivered Kt/V. *Am J Kidney Dis.* 1999;33:1131–1135.
47. Elisaf M, Bairaktari H, Tziallas C, Germanos N, Siamopoulos KC. Atherogenic lipid and lipoprotein parameters in hemodialysis patients. *Dial Transplant.* 1995;24:642–650.
48. Schreiber B. Levocarnitine and dialysis: a review. *Nutr Clin Pract.* 2005;20: 218–243.
49. Russell GI, Davies TG, Walls J. Evaluation of the intravenous fat tolerance test in chronic renal disease. *Clin Nephrol.* 1980;13:282–286.
50. Skipper A, Marian M. Parenteral nutrition. In: Gottschlich M, Matarese L, Shronts E, eds. *Nutrition Support Dietetics Core Curriculum.* 2nd ed. Silver Springs, Md: American Society for Parenteral and Enteral Nutrition; 1993:118.
51. Fuhrman M. Micronutrient assessment in long-term home parenteral nutrition patients. *Nutr Clin Pract.* 2006;21: 566–575.
52. Wolk R. Micronutrition in dialysis. *Nutr Clin Pract.* 1993;8:267–276.
53. Kelly MP, Kight M, Rodriguez R, Castillo S. A diagnostically reasoned case study with particular emphasis on B6 and zinc imbalance directed by clinical history and nutrition physical examination findings. *Nutr Clin Pract.* 1998;13:32–39.
54. Fouque D, Vennegoorz M, Ter Wee P, et al. EBPG guideline on nutrition. *Nephrol Dial Transplant.* 2007;22 (suppl 2):ii45–ii87.
55. Kalantar-Zadeh K, Kopple JD. Trace elements and vitamins in maintenance dialysis patients. *Adv Ren Replace Ther.* 2003;10:170–182.
56. Hsieh YY, Shen WS, Lee LY, et al. Long-term changes in trace elements in patients undergoing chronic hemodialysis. *Biol Trace Elem Res.* 2006;109:115–122.
57. Wolk R, Moore E, Foulks C. Renal disease. In: Gottschlich M, DeLegge M, Mattox T, Mueller C, Worthington P, eds. *The A.S.P.E.N. Nutrition Support Core Curriculum-A Case-Based Approach – The Adult Patient.* Silver Spring, Md: American Society for Parenteral and Enteral Nutriton; 2007:575–598.
58. Kraft M, Btaiche I, Sacks G. Review of the refeeding syndrome. *Nutr Clin Pract.* 2005;20:625–633.

Correction

Sharp-eyed readers noticed several errors in Table 6 in the article "Transitioning Nutrition Management of the Preterm Neonate from the Hospital to the Home" in the June 2007 issue of *Support Line.* A corrected version of the table follows:

Table 6. Common Vitamin and Mineral Supplementation for Discharge (23)

Product	Poly-Vi-Sol	Poly-Vi-Sol With Iron	Tri-Vi-Sol	Tri-Vi-Sol With Iron	Fer-In-Sol	Water-soluble Vitamin	Calcium*	Phosphorus**	Vitamin D***
Dosage	1 mL	1 mL	1 ml	1 mL	0.6 mL	1 mL	1 mL	1 mL	1 mL
Vitamin A (IU)	1,500	1,500	1,500	1,500	-	1,500	-	-	-
Vitamin D (IU)	400	400	400	400	-	400	-	-	8,000
Vitamin K (mg)	-	-	-	-	-	0.1	-	-	-
Vitamin E (IU)	5	5	-	-	-	40	-	-	-
Vitamin C (mg)	35	35	35	35	-	45	-	-	-
Thiamin (mg)	0.5	0.5	-	-	-	0.5	-	-	-
Riboflavin (mg)	0.6	0.6	-	-	-	0.6	-	-	-
Biotin (mcg)	-	-	-	-	-	15	-	-	-
Niacin (mg)	8	8	-	-	-	6	-	-	-
Vitamin B6 (mg)	0.4	0.4	-	-	-	0.6	-	-	-
Vitamin B12 (mcg)	2	2	-	-	-	4	-	-	-
Iron (mg)	-	10	-	10	15	-	-	-	-
Calcium (mg)	-	-	-	-	-	-	23	-	-
Phosphorus (mg)	-	-	-	-	-	-	-	127	-
Zinc (mg)	-	-	-	-	-	5	-	-	-

Poly-Vi-Sol® with or without Iron, Tri-Vi-Sol® with or without Iron, and Fer-In-Sol® are made by Mead Johnson and Company, Evansville, Ind.

*Elemental calcium in the form of calcium glubionate

**Elemental phosphorous in the form of Fleets Phosphosoda orally (Fleet Company, Lynchburg, Virginia)

***Vitamin D in the form of ergocalciferol.

The Evidence Analysis Library and Critical Illness Guidelines: An Introduction to the Process

Ainsley M. Malone, MS, RD, CNSD

Abstract

Evidence analysis is an integral aspect of evidenced-based medicine, a movement initiated in the past decade. The American Dietetic Association (ADA) recognized the need for dietetic practice to move in this direction and subsequently established and developed a rigorous process for evidence analysis. Their commitment to and support for this approach to research led to the creation and release of the Evidence Analysis Library (EAL). Dietitians in Nutrition Support (DNS) conducted initial evidence analysis work and subsequently merged with ADA's efforts through creation of the critical illness work group. This article reviews the ADA EAL and the Critical Illness (CI) Guidelines available to ADA and DNS members.

Introduction

The philosophy of evidenced-based practice was introduced to the medical community in 1981 at a small Canadian University (1). This approach centered on teaching clinicians how to appraise the medical literature critically and apply this knowledge to patient care problems. The term "evidence-based medicine" was described in a landmark article in the *Journal of the American Medical Association* in 1992 and formed the beginning of what is now common to all aspects of medicine (2).

Evidence-based medicine has had a substantial impact on dietetic practice for almost a decade. In 1998, Porter and Matel (3) offered one of the first reviews of evidence-based medicine, describing the need to change from experiential-based decision making to that which is based on the best available evidence. In 2000, the ADA published a toolkit for dietetics professionals on developing evidence-based guides for practice. (4) Subsequently, Medical Nutrition Therapy Evidence-based Guides for Practice for various disease states were published and made available to members. (Available at http://www.eatright.org/cps/rde/xchg/ada/hs.xsl/education_3876_ENU_HTML.htm.)

Recognizing the significant impact that evidence-based practice would have for its members, the ADA initiated a commitment in 2004 to support this decision-making approach by appointing an Evidence-based Practice Committee (5). This commitment received both financial funding and incorporation of support throughout ADA's entire program of work. In September 2004, the EAL was made available to ADA members with the release of evidence analysis for Disorders of Lipid Metabolism. Since that beginning, the growth of the EAL has been exponential, with the inclusion of 24 current or ongoing projects (Table 1), Evidence-based Nutrition Practice Guidelines for several disease entities, and an Evidenced-based Toolkit. This article introduces the structure and process of the EAL and specifically the critical illness (CI) section. Practical application of the EAL will be explored in a subsequent article.

(Continued on next page)

Table 1. Current ADA Evidence Analysis Library Projects

- Adult Diabetes 1 and 2 (revision)
- Adult Weight Management*
- Childhood Overweight
- Chronic Kidney Disease (revision)†
- Chronic Obstructive Pulmonary Disease†
- Critical Illness*‡
- Disorders of Lipid Metabolism (Hyperlipidemia revision)* ‡
- Energy Expenditure: Measurement Versus Estimation
- Fiber
- Gestational Diabetes (revision)†
- Gluten Intolerance/Celiac
- Heart Failure†
- HIV/AIDS
- Hydration
- Hypertension†
- Non-nutritive Sweetener
- Nutrition in Athletic Performance
- Nutrition Care in Bariatric Surgery
- Nutrition Counseling
- Oncology†
- Pediatric Weight Management*
- Spinal Cord Injury and Nutrition
- Unintended Weight Loss
- Vegetarian Nutrition

*Guidelines currently available on the EAL.
†Work groups with evidence-based guidelines under development.
‡Toolkit available or under development

The EAL: Purpose and Process

Evidence-based dietetics practice can be defined best as "the use of systematically reviewed scientific evidence in making food and nutrition practice decisions" and is achieved by integrating the best available evidence with professional expertise and client values to improve outcomes (6). This working definition of evidence-based dietetics practice, as developed by the ADA Evidence-based Practice Committee, guides the process of evidence analysis and publication on the EAL. The potential benefits of using an evidence-based process are significant (Table 2) (7). Providing practitioners with evidenced-based answers to their clinical questions can only enhance their practice. Using evidence-based tools not only is empowering, but it leads to increased credibility with other health-care professionals.

Steps in the ADA Evidence Analysis Process

Table 3 outlines the steps in the evidence analysis process used by ADA. This procedure was developed by the Quality Management Committee and is based on the systems developed by the Institute of Clinical Systems Improvement (8). The first step in the process, selecting a topic and establishing an expert work group, is managed by the Evidence-based Practice Committee, which prioritizes all topics to be considered for the EAL and appoints an expert work group comprised of a balance of practitioners and researchers who are knowledgeable in the specific topic area. Step 2 in the process focuses on the development of the precise questions to be answered by evidence analysis, which are specific to a defined area of practice. Questions are formulated using an outcomes-based approach and follow a model incorporating the following (PICO) components: population, intervention, comparison, and outcome (9). For example, with respect to CI, the question may be, "What outcome do we expect if enteral nutrition (EN) rather than parenteral nutrition (PN) is provided to critically ill patients?" Expert work group members are integral to question development and formulation. Step 3 begins the search for evidence by establishing exclusion and inclusion criteria for research so that the most appropriate research is used for answering the specific question. An extensive and systematic literature search using multiple MeSH (medical subject heading) terms and a variety of databases is conducted in Step 4. This search results in the creation of a sort list, a list of included and excluded research articles for the specific question, which is presented to the work group for approval. Selected questions asked by the work group in final article decision making include: How well does the research answer the specific question being asked? Does the research involve the appropriate population? Step 5 involves the critical appraisal of each included article for the question. A trained evidence analyst reviews each article, completes an extensive worksheet, and evaluates the article quality via a defined quality rating system (10). Step 6 follows the appraisal and involves organizing and summarizing the articles into an overview table and a narrative evidence summary, which is approved by the work group. A conclusion statement, based on the reviewed literature, is drafted in Step 7 and is discussed, reviewed, and approved by the work group. In Step 8, the work group assigns a grade to the statement based on the EAL evidence grading system (Table 4). The final step in the evidence analysis process is the culmination of all the previous steps: publishing the final conclusion statements, evidence summary, overview table, and evidence worksheets on the EAL.

As previously outlined, a large number of medical conditions currently are undergoing evidence analysis. When most of the defined questions have been answered within a project, members of the work group convene to develop guidelines for using the evidence in practice. This provides a transition from what the evidence outlines to a course of action that can be taken by the practitioner based on the evidence. Within the guidelines are recommendations or "action statements" that guide the practitioner; an algorithm providing a step-by-step flow for use; and links to the appropriate conclusion statements, evidence summaries, and worksheets for the disease or condition. Currently, Evidence-based Nutrition Practice Guidelines have been created or are under development for the projects highlighted in Table 1. Following release of a guideline, a toolkit is

Table 2. Potential Benefits of the Use of Evidence-based Practice (7)

- Improves the quality of dietetic practice through promoting practices that support the best outcomes
- Decreases the often wide variations in practice
- Reduces the gap between what has been demonstrated through research and what occurs via anecdotal evidence and expert opinion
- Allows the clinician to take advantage of rapidly expanding medical knowledge
- Saves time

Table 3. Steps in the ADA Evidence Analysis Process

1. Select topic and appoint expert work group.
2. Define questions using the PICO format.
3. Determine inclusion and exclusion criteria.
4. Conduct literature review for each question.
5. Analyze articles and provide critical appraisal.
6. Complete overview table and evidence summary.
7. Develop conclusion statement.
8. Achieve consensus on evidence grade.
9. Publish to online EAL.
10. Develop toolkit for application.

created to help apply the guidelines and offer multiple resources for practitioners. Some of the components of the toolkits include case studies, monitoring forms, intervention and encounter forms, and outcome tools. Currently, a toolkit is available for the Disorders of Lipid Metabolism, and the release of a CI Toolkit is expected in the fall of 2007.

The evidence analysis process used by the ADA for the EAL is highly rigorous and has been praised by other organizations for its thoroughness and quality. The process was adapted by the United States Food and Drug Administration to assess the type of qualified health claims that can be placed on food labels. In addition, the process has been recognized by The Joint Commission as exemplary in bringing the best research to practice (9). Users of the EAL should feel extremely confident with the results offered.

Critical Illness Work Group and Guidelines

In 2002, the leaders of DNS recognized the need for an evidenced-based evaluation of multiple practices related to the use of PN and EN. This need was identified while completing a 2000 update of EN protocols. DNS formed a committee of seven nutrition support dietitians to conduct an evidence analysis of multiple key questions pertaining to enteral feedings (Table 5) (11). Initially, the committee was staffed and financially supported entirely by DNS, but once the ADA process was developed and underway, the work group merged with ADA's process to contribute both financial and professional expertise. To date, there are approximately 62 conclusion statements and 49 evidence summaries involving 9 selected topics within the CI disease entity on the EAL. The CI Evidence-based Nutrition Practice Guideline offers 19 recommendations encompassing evidence from primarily the CI analysis but also selected evidence from the Adult Weight Management work group. Table 6 provides a list of the recommended topics included in the CI guidelines.

(Continued on next page)

Table 4. Grading System for Concluding Statements: Narrative Explanation of Grades (8,9)

- **Grade I: Good** — The evidence consists of results from studies of strong design for answering the question addressed. The results are both clinically important and consistent with minor exceptions at most. The results are free of serious doubts about generalizability, bias, and flaws in research design. Studies with negative results have sufficiently large sample sizes to have adequate statistical power.
- **Grade II: Fair** — The evidence consists of results from studies of strong design answering the question addressed, but there is uncertainty attached to the conclusion because of inconsistencies among the results from different studies or because of doubts about generalizability, bias, research design flaws, or adequacy of sample size. Alternatively, the evidence consists solely of results from weaker designs for the questions addressed, but the results have been confirmed in separate studies and are consistent with minor exceptions at most.
- **Grade III: Limited** — The evidence consists of results from a limited number of studies of weak design for answering the questions addressed. Evidence from studies of strong design is either unavailable because no studies of strong design have been done or because the studies that have been done are inconclusive due to lack of generalizability, bias, design flaws, or inadequate sample sizes.
- **Grade IV: Expert Opinion Only** — The support of the conclusion consists solely of the statement of informed medical commentators based on their clinical experience, unsubstantiated by the results of any research studies.
- **Grade V: Not Assignable** — There is no evidence available that directly supports or refutes the conclusion.

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Table 5. Enteral Feeding Questions Developed for Evidence Analysis by DNS Leaders

1. What is the effect of enteral versus parenteral feeding on infectious complications, cost, length of hospital stay (LOS), and mortality?
2. Does the timing of enteral feeding influence infectious complications, LOS, or mortality?
3. Does the placement of an enteral feeding tube tip in the gastric versus postpyloric position affect gastric residual volume or reflux, aspiration pneumonia, cost, LOS, or mortality?
4. What monitoring criteria should be used for enteral feeding management?
5. Does the amount of enteral formula actually delivered influence infectious complications, cost, LOS, ventilator days, or mortality?
6. Does the use of blue dye aid in the detection of aspiration or influence mortality?

At present, additional questions pertaining to weight, caloric and protein delivery, and their relation to clinical outcomes is underway.

Summary

Evidenced-based dietetic practice is essential for all practitioners. Not only does it provide an objective approach to decision making, but it improves the credibility of the RD within the heath-care team. The ADA EAL employs a highly rigorous process to analyze existing literature, providing the practitioner with a summary of what the evidence demonstrates for many different medical conditions. The CI guidelines provide recommendations to guide the nutrition support dietitian in making practice decisions for critically ill patients. They contribute to the nutrition care model in which the dietitian takes a patient's individual needs, research, and guidelines into account in managing nutrition therapy. Using the best available evidence can enhance the care provided to patients, with the ultimate goal of improving patient outcome.

Table 6. Recommendation Topics Included in the Critical Illness Guidelines

- Enteral versus parenteral nutrition
- Timing of enteral nutrition and critical illness
- Immune-enhancing enteral nutrition and critical illness
- Gastric versus small bowel feeding tube placement
- Blue dye use and critical illness
- Monitoring criteria in critical illness
- Monitoring delivery of energy in critical illness
- Blood glucose control
- Gas (oxygen and carbon dioxide) collection devices
- Impact of the thermic effect of feeding on resting metabolic rate
- Effects of different length rest periods (a specific amount of rest time prior to a measurement) on resting metabolic rate
- Impact of environmental factors on resting metabolic rate measurement: physical comfort/posture
- Steady-state measurement conditions and number of measurements in a 24-hour period
- Respiratory quotient as a method to detect measurement error
- Determination of resting metabolic rate

Acknowledgment: The author wishes to recognize the significant effort of DNS leaders who initiated the early evidence analysis work: Pam Charney, Charlene Compher, Mary Hise, Kendra Kattelmann, Mary Russell, and Milton Stokes. Without their dedication and commitment, the large body of topics within the critical illness disease entity on the EAL would not exist.

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References

1. Guyatt G. Preface. In: Guyatt G, Rennie D, eds. *Users' Guides to the Medical Literature: A Manual for Evidence Based Clinical Practice.* Chicago, Ill: AMA Press; 2002:xiii–xvi.
2. Evidence-Based Medicine Working Group. Evidence-based medicine: a new approach to the teaching of medicine. *JAMA.* 1992;268:2420–2425.
3. Porter C, Matel JL. Are we making decisions based on evidence? *J Am Diet Assoc.* 1998;98:404–407.
4. Splett P. *Developing and Validating Evidence-Based Guides for Practice: A Toolkit for Dietetics Professionals.* Chicago, Ill: The American Dietetic Association; 2000.
5. Reeves RS. Building the structure of dietetics practice. *J Am Diet Assoc.* 2005;105:1849.
6. American Dietetic Association. About Guidelines. *American Dietetic Association Evidence Analysis Library.* Available at: http://www.adaevidencelibrary.com/category.cfm?cid=10&cat=0. Accessed June 2007.
7. Gray GE, Gray LK. Evidence-based medicine: applications in dietetic practice. *J Am Diet Assoc.* 2002;102:1263–1272.
8. Greer N, Mosser G, Logan G, Halaas G. A practical approach to evidence grading. *Jt Comm J Qual Improve.* 2000;26:700–712.
9. American Dietetic Association. What is the ADA evidence analysis process? *American Dietetic Association Evidence Analysis Library.* Available at: http://www.adaevidencelibrary.com/category.cfm?cat=1330&cid=7. Accessed May 2007.
10. American Dietetic Association. *American Dietetic Association Evidence Analysis Work Groups.* Available at: http://www.adaevidencelibrary.com/files/File/Work_Group_responsibilities.pdf. Accessed May 2007.
11. Kattelmann KK, Hise M, Russell MK, Charney PA, Stokes M, Compher C. Preliminary evidence for a medical nutrition therapy protocol: enteral feedings for critically ill patients. *J Am Diet Assoc.* 2005;106:1226–1241.

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Nutrition Management of the Geriatric Patient

Maryann King, MPH, RD, CNSD Elizabeth Emery, MS, RD, CNSD

Abstract

Nutrition can be one of the most important factors contributing to the aging process. Approximately 85% of the older population (65 years of age and older) have at least one chronic condition that has been documented to benefit from nutrition intervention (1). One of the most challenging aspects of providing nutrition to the elderly is determining their nutritional status because aging can affect many of the parameters used to assess such status. The health professional must conduct a thorough nutrition assessment in the geriatric patient, including appraisal of physical appearance, oral health, social and environmental situations, potential physical and psychologic disabilities, medical and drug history, and hematologic and immunologic data (2). Every attempt should be made to meet the nutrition needs of the elderly through oral intake. Recent studies have raised doubts about the long-term benefits of and concerns about the overuse of percutaneous endoscopic gastrostomy (PEG) tubes in the geriatric population. Better guidelines are needed to identify appropriate candidates for enteral and parenteral nutrition support in this population.

Demographics of Aging

At the beginning of the 20th century, the average life expectancy was approximately 42 years of age. Life expectancy has nearly doubled in the past century and has come close to tripling during the course of human history (3). By the year 2030, more than 70 million Americans will be older than age of 60 years, and the number of centenarians is projected to increase from 37,000 in 1990 to approximately 131,000 in 2030 (4). Clinicians need to be acutely aware of the health-care and nutrition needs of this growing population. Optimal nutritional status is important for good health at any age, but it requires particular attention in the elderly. A substantial proportion of older Americans are at risk for malnutrition because of age-related declines in organ systems, inability to acquire food and prepare nutritious meals, medication-induced deficiencies, and the impact of chronic disease (2). This article highlights physiologic changes that affect nutritional status and addresses challenges in nutrition assessment and support in this growing population.

Nutrition Assessment

It is estimated that 5% to 10% of community-dwelling older adults, 17% to 65% of the elderly in acute care hospitals, and 5% to 59% of those in long-term care institutions are undernourished (5). One of the more challenging aspects of providing appropriate nutrition intervention to elderly adults is determining their nutritional status. Often, pathologic conditions are mistaken for aspects of normal aging, by both older adults and health-care professionals, including malnutrition, symptoms of which include muscle wasting and weight loss. Failure to thrive, a term typically used to describe children who have delayed physical growth, is applied to the elderly to indicate deterioration in functional status disproportionate to their disease burden (6). The causes of failure to thrive are multifactorial and include undernutrition, loss of muscle mass, problems with balance and endurance, declining cognition, and depression. Aging affects many of the anthropometric, biochemical, and hematologic parameters commonly used to assess nutritional status in the younger adults.

Aging is associated with changes in skin elasticity, hydration, and compressibility as well as changes in subcutaneous and connective tissues that can alter the relationship of skinfold thickness measurements to body composition (7). Such changes affect the accuracy of anthropometrics (7). Another significant limitation of anthropometry in the older population is the lack of appropriate standards with which to compare results; standards are derived from measurements compiled from younger adults and do not adjust for age-related physiologic changes. Therefore, anthropometrics are not considered accurate in the elderly population (8). The reliability of body mass index (BMI) in the elderly also has been questioned because of concerns related to changes in height and body composition (8).

Aging successfully depends on a variety of factors, including genetic inheritance, health habits, lifestyle, environmental factors, chronic and acute disease, and access to health care. Research has shown that behaviors such as eating a healthy diet, being physically active, and not using tobacco are more influential than genetic factors in helping individuals avoid the deterioration associated with aging (9).

Nutrition assessment in the elderly should include appraisal of physical appearance, including height, weight, and knee height (if height is not available); oral health; social and environmental situations; physical and psychological disabilities; medical history; diet history; and evaluation of biochemical, hematologic, and immune function (10).

Physiologic Changes Associated With Aging

Muscular

The human aging process includes progressive changes in body composition, metabolism, physiologic function, physical activity, mental status, food intake, frequency of disease, and the ability of the body to respond to these changes. Such changes are both progressive and integrated. For example, sarcopenia, the age-associated loss of skeletal muscle mass, muscle strength, and muscle efficiency, has been linked to declines in physical activity, motor neuron function, and protein synthesis as well as concentrations of hormones, insulin-like growth factor (IGF-1), testosterone, estrogen, growth hormones (GHs), and serum albumin (11).

GH stimulates cell growth and protein synthesis, which increases lean body mass. One study found a 29% to

(Continued on next page)

70% lower secretion of GH over a 24-hour period in elderly men compared with younger men (12). Such a decrease in GH may contribute to the loss of lean body mass. Concentrations of IGF-1, which is released by the liver in response to GH, have been shown to decline with aging. Rudman and colleagues (13) randomized 21 healthy men ages 61 to 81 years of age, who had low plasma IGF-1 concentrations, into two groups and obtained baseline measures for 6 months. They then provided one group (n=12) with 0.03 mg/kg body weight of biosynthetic human GH subcutaneously three times per week for 6 months. In the subsequent 6-month period, the treatment group exhibited an 8.8% increase in lean body mass and a 14.4% decrease in total body fat over baseline levels compared with the nontreatment group. There was no statistically significant improvement in muscle strength or systemic endurance despite improvements in body composition (13). The treatment group also had mean IGF-1 values increase into the range usually seen during the teenage years; concentrations in the placebo group remained constant throughout the entire study period (13). This study extends the understanding of potential benefits of GH administration on body composition, but the use of GH has not become standard practice. GH can affect carbohydrate metabolism adversely, producing hyperinsulinemia, glucose intolerance, and diabetes mellitus (14). The risk/benefit ratio has not been established in older adults (14).

Cardiovascular

Cardiovascular decline has been associated with the aging process, but the degree of change varies among individuals (15). Nutrition interventions now are used as both initial and adjuvant therapies for the treatment of cardiovascular disease (CVD) risk factors, including hypertension, diabetes, and lipid disorders. However, fewer than 50% of adults older than age 65 report receiving nutrition-related guidance from their health-care providers (16). Diseases of the heart and blood vessels are, by far, the most important cause of morbidity and mortality among elderly individuals (17). Approximately 84% of CVD deaths occur in people older than age 65 (18). Primary efforts to prevent CVD should begin with altering lifestyle habits during middle age. Many experts believe that these efforts should begin considerably earlier (19). Prevention activities include maintaining a healthy body weight, detecting and treating hypertension, maintaining a blood lipid profile consistent with minimizing risk, smoking cessation, and increased physical activity. Most of these risks are affiliated with dietary factors (16). However, calorie restriction must be undertaken carefully in this patient population that is already vulnerable to malnutrition. When trying to achieve weight reduction and maintenance, it may be beneficial to encourage increased physical activity as tolerated. Any dietary modification in older individuals should encompass a careful assessment to determine both the nutritional adequacy of the diet and the level of intake of nutrients to be modified.

Renal

Renal function declines with age; approximately 40% of the nephrons become sclerotic between the ages of 25 and 85 years (20). Such loss of renal mass is primarily from the cortex and is vascular in origin. The progression does not affect everyone, and it appears to be due to a combination of environmental and genetic factors (21). The changes are accelerated in individuals who have diabetes, hypertension, dyslipidemia, and atherosclerotic disease (21). Renal tubular function also declines with advancing age. The ability to conserve sodium and excrete hydrogen ions falls, resulting in diminished capacity to regulate fluid and acid-base balance. Dehydration becomes a particular problem because the aging kidney does not compensate for nonrenal losses of sodium and water by the usual mechanisms of increased renal sodium retention, increased urinary concentration, and increased thirst. The changes are believed to be due to a decline in the activity of the renin-angiotensin system and decreased responsiveness to antidiuretic hormone (20).

Dehydration is a major problem in the older adult, especially persons older than 80 years of age (Table). Phillips and associates deprived healthy young men and older men of water for 24 hours (22). After fluid deprivation, the two groups had identical weight loss and similar changes in plasma volume, but the older men had an increase in plasma sodium concentration and osmolality. During rehydration, the older group did not drink enough water to dilute body fluids and were not markedly thirsty; the younger men did consume adequate fluids to correct fluid status. In general, fluid needs of the older adult can be met with 30 mL/kg of body weight per day (22).

Gastrointestinal

Some changes in structure and function of the gastrointestinal tract occur with age, but they do not seem to be clinically significant. Gastric secretion does not decrease solely due to advanced age, as was believed in the early 1990s (23). Gastric acid secretion may be affected by gastritis commonly caused by *Helicobacter pylori* (24). Gastrointestinal problems tend to localize in the proximal and distal portions of the gut. The primary organic causes of dyspepsia in the elderly patient are gastroduodenal ulcer, atypical gastroesophageal reflux disease, and gastric cancer (25). Up to 60% of patients who have dyspepsia have no definite organic explanation

Table. Risk Factors for Dehydration in the Elderly

- Anorexia
- Laxative abuse
- Diuretic abuse
- Disability
- Confinement to chair or bed
- Confusion/mental impairment
- Diarrhea
- Vomiting
- Hemorrhage
- Incontinence
- Unconsciousness
- Renal insufficiency
- Dependency on nutrition support
- Chronic disease
- Polypharmacy
- Presence of chronic infections
- Swallowing impairment
- Physical anomalies

and are classified as having functional dyspepsia (25).

A decrease in calcium absorption has been attributed to decreased renal production of 1,25-hydroxycholecalciferol as well as a decrease in the amount and/or sensitivity of intestinal mucosal calcium-binding proteins (26).

Malnutrition that is unexplained by diet factors alone can be secondary to malabsorption due to bacterial overgrowth (26). The overgrowth of microflora disturbs intraluminal digestion and mucosal function, potentially resulting in malabsorption of fat, protein, carbohydrate, electrolytes, and vitamin B_{12}. Malnutrition due to steatorrhea and macrocytic anemia due to vitamin B_{12} deficiency frequently develop. The clinical symptoms of bacterial overgrowth include abdominal pain, bloating, diarrhea, and weight loss. Treatment involves antibiotic administration in 2-week courses that may need to be repeated at varying intervals (26). Antibiotics may cause diarrhea. *Saccharomyces boulardii*, a nonpathogenic yeast that is widely commercially available, appears to reduce rates of antibiotic-associated diarrhea significantly (27). Meta-analysis found a reduction in antibiotic-associated diarrhea from 17.2% in controls to 6.7% of the *S boulardii*-treated patients. A narrative review has summarized recent studies of probiotics for the treatment of diarrhea (28). The authors concluded that *Lactobacillus*, *Bifidobacterium*, and *Saccharomyces* sp were the best studied and had the greatest potential for preventing or treating antibiotic-associated diarrhea, acute rotavirus-induced diarrhea, and traveler's diarrhea. However, the studies were not consistent, which may reflect methodology, type or severity of diarrhea, or type or biologic availability of the probiotic (28).

The prevalence of constipation appears to increase among the elderly. The cause is multifactorial and includes a sedentary life style, poor diet, dehydration, anorectal and colonic pathology, systemic illness, and medication (29).

Hepatic

With age comes a decrease in the number of hepatocytes and the overall weight and size of the liver. In advanced age, the liver becomes disproportionately small, most likely because of decreased numbers of hepatocytes and systemic disease (30). Hepatic blood flow can decrease as much as 40%, which impairs the disposition of drugs. Concentrations of albumin, a product of hepatic synthesis, frequently are reduced in older adults (30). Albumin values also can be normal or high in elderly patients who have chronic undernutrition or dehydration.

The most common manifestation of altered hepatobiliary function in old age is the increased incidence of gallstones and gallstone-related complications. The prevalence of gallstones rises steadily with age; biliary stones have been documented in up to 80% of nursing home residents older than the age of 90 years (31).

Other Physiologic Changes

The decline in immune competence that accompanies aging is characterized by an increased susceptibility to infections, an increase in autoantibodies and monoclonal immunoglobulins, and an increase in tumorigenesis (32). The decline in immune function may not be apparent in an unchallenged state. For example, there is no decline in neutrophil count with age, but the ability of the bone marrow to increase neutrophil production in response to infection may be impaired (32). Elderly patients who have major infections frequently have normal white blood cell counts, but the differential cell count reveals a profound shift (32).

Oral health can be greatly affected by the aging process. Chewing and swallowing problems are the most easily recognized causes of nutrition failure, often resulting in poor oral intake. Approximately 25% of adults older than age 60 are edentulous (33). Individuals who have missing teeth or dentures tend to choose softer foods and avoid fresh fruits, vegetables, and whole grains. Other factors that reduce oral intake are the frequent drug-induced alterations of taste, nausea, and disturbed mood or attention (33). The aging process does not decrease the amount of salivary flow; recent evidence indicates that the diminished salivary flow often noted in studies of the elderly is due to pathologic conditions or pharmacologic effects of medications (34). The cumulative effects of aging, disease, and trauma contribute to the wide variety of oral health problems that are prevalent in the aging population. There is a strong relationship between oral health status, food selection, chewing efficiency, and ability to swallow, all of which affect the nutritional status of the elderly (35). Other risks factors for malnutrition include inadequate physical activity, physical disabilities, numerous medications, social isolation, poverty, and depression (35).

Oral Nutrition Support in the Elderly

The multitude of factors that can compromise nutritional status in the elderly can lead to geriatric failure to thrive, a syndrome that includes decreased appetite, weight loss, poor nutrition, and inactivity often accompanied by dehydration, depression, impaired immunity, and low cholesterol concentrations (36,37). Low BMI and unintentional weight loss in the elderly has been associated with increased mortality in a number of studies (36,38–40). Energy intake is well known to decline during aging, with some studies showing calorie consumption falling by as much as 1,200 kcal/day in men and 800 kcal/day in women (41,42). Due to decreases in energy expenditure that occur with aging, a reduction in energy intake may be desirable, but decreased food intake generally is accompanied by reductions in nutrient intake. Furthermore, although the recommended dietary allowance (RDA) for protein is 0.8 g/kg for adults of all ages, it is believed that elderly persons need at least 1.0 g/kg/day to maintain nitrogen balance (43). Even if the RDA of 0.8 g/kg is used, 25% to 40% of persons aged 65 and older consume less than this amount (44).

Providing a diet that is nutritious, highly palatable, easy to eat, and culturally appropriate is important in maintaining sufficient energy and protein intake in the elderly. The addition of oral protein and energy supplements, in the form of liquid supplements or fortified foods, should be attempted when dietary intake is inadequate. Two systematic reviews by Milne and associates (45,46) involving more than 9,000 elderly people suggested that protein and energy

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supplementation can be helpful in promoting weight gain and may have beneficial effects on morbidity and mortality for those who are undernourished at baseline. Some authors have noted a decrease in oral food intake in response to energy-dense supplements (47); serving supplements between meals, rather than with meals, may be more effective in increasing energy consumption (48). Although not well studied, increasing the energy and nutrient density of foods served at meals via fortification may be a solution to this problem.

Micronutrient Deficiencies

Vitamins may play a role in prevention of the pathogenesis of most chronic diseases of aging, such as decreased cognitive function, cardiovascular diseases, and cancer. In addition to the potential impact on energy and protein intake, aging can alter micronutrient status. Vitamin and mineral deficiencies are likely to result from inadequate intake as well as alterations in absorption and metabolism (49,50). Vitamin B_{12}, vitamin D, and calcium are among the micronutrients that may be deficient in the elderly and are reviewed in this article.

Vitamin B_{12} deficiency occurs in about 20% of the general population in industrialized countries (51). Vitamin B_{12} is obtained exclusively from animal and dairy foods. Because of poor dentition, taste alterations, and economic factors, intake of meats is often low in the elderly. In addition, adequate gastric acid is required for the liberation of vitamin B_{12} from animal proteins before it is attached to intrinsic factor. Atrophic gastritis, which occurs in 40% to 50% of elderly persons older than age 80 years, and the chronic use of acid-suppressing drugs poses a significant obstacle to the absorption of vitamin B_{12} (49,51). The Institute of Medicine recommends that most of the RDA for vitamin B_{12} for adults older than age 50 years be met through foods fortified with vitamin B_{12} or B_{12} supplements (52). Deficiencies of vitamin B_{12} in the elderly can contribute to anemia and neuropathies and may be related to dementia. (51,53,54). Recent findings suggest that fortification of the diet with folic acid to reduce neural tube defects could have a negative effect on elderly persons with vitamin B_{12} deficiency. Poor vitamin B_{12} status combined with high folate intake from fortified foods and supplements has been associated with cognitive impairment and declines in cognitive function in the elderly (53–56). This effect on cognition is not seen when high folate intake occurs in the presence of normal B_{12} status; in fact, a combination of high folate intake and normal B_{12} status appears to be protective against cognitive impairment (54). It has been suggested that elderly persons taking folic acid supplements or exceeding the upper intake level for folate from supplements and fortified foods also should take vitamin B_{12} (57).

Vitamin D is synthesized from cholesterol in the skin and obtained in the diet from fish and fortified foods, including dairy products and cereals. Dietary intakes are commonly low in the United States (58,59), and cutaneous production decreases with age. Limited sun exposure due to institutionalization and liberal use of sunscreens also contributes to reduced vitamin D synthesis. Although it is not the physiologically active form, 25-hydroxyvitamin D is the metabolite that is used to assess vitamin D in the general population. Nearly 50% of independently living elderly Americans in northern latitudes have vitamin D insufficiency, as evidenced by low serum 25-hydroxyvitamin D concentrations (58).

The adequate intake for vitamin D for adults older than age 70 years is 15 µg/day (600 IU), an increase over the recommendation of 10 µg/day (400 IU) for adults ages 51 to 70 years old (60). Higher intakes, up to 20 µg/day (800 IU), may be required for optimal bone heath in the elderly (61). Vitamin D deficiency is known to contribute to osteoporosis (62). In addition, a recent meta-analysis suggested that vitamin D supplementation can reduce fracture risk independently of its effect on bone strength by reducing falls through improved muscle strength (63). Thus, vitamin D may contribute to reduced risk of fracture by enhancing both bone strength and agility. Supplements often are necessary to achieve adequate vitamin D status in this population.

Calcium requirements also increase with age. The recommended adequate intake for calcium rises from 1,000 mg for adults ages 18 to 50 years to 1,200 mg for both ages 51 to 70 and older than age 70, with some sources recommending up to 1,500 mg/day for postmenopausal women not taking estrogen replacement therapy (60,64). Deficiencies of vitamin D or calcium can lead to secondary hyperparathyroidism and subsequent bone loss. Osteoporosis and fractures are major public health problems that are expected to grow as the elderly population expands (61). The average intake of calcium in the United States is only 600 mg/day, and intakes in the elderly are even lower (59,61). Calcium supplementation has been shown to be effective in reducing bone loss in elderly women (61). A convenient way to meet both calcium and vitamin D needs is a combination supplement that contains calcium, vitamin D, and other nutrients that promote bone health. Calcium supplements should be taken in divided doses not exceeding 500 mg, preferably with meals.

Other micronutrients, including zinc, iron, folic acid, vitamin C, and vitamin A, can be of particular importance in the frail elderly, and their use has been reviewed elsewhere (49,50,59,64). Dietary intakes and potential deficiencies should be assessed on an individual basis.

Enteral Nutrition Support in the Elderly

When the gut is working, but nutrition needs cannot be met through an oral diet or supplements, enteral nutrition may be the next step. Although a common intervention, the provision of enteral feedings to the elderly has not been without controversy, particularly for elderly patients with dementia and those who no longer are able to make their own medical decisions for other reasons. In 1999, Finucane and associates (65) published a literature review attempting to identify if enteral feedings in patients who had advanced dementia could change outcomes. Specifically, they evaluated whether enteral feedings decreased the risk of aspiration pneumonia, pressure ulcers, or infection and whether tube feedings could improve function, survival, or palliation. They concluded that no data suggested clinical improvement, risks were substantial, and "the widespread practice of tube

feeding should be carefully reconsidered" (65). Three months later, Gillick published an article supporting this opinion (66). These authors raised important questions and were part of an early debate that sparked substantial professional interest in the topic.

No prospective randomized clinical trials have examined the question of whether elderly patients benefit from enteral feedings, and it is unlikely that ones will be performed due to ethical reasons. However, retrospective observational studies have compared outcomes in patients with and without feedings. In these studies, risk of aspiration and pressure ulcer development was not reduced with enteral nutrition (67). Patients with dementia experienced no apparent improvement in ability to communicate or perform activities of daily living (68). In addition, tube feedings were not associated with prolonged survival. In a small study of 41 candidates for enteral feedings, 23 received PEGs and 18 refused feedings (69). No difference in survival between these groups was seen, with the median survival for the PEG patients being 59 days and 60 days for those without tube feeding. An earlier study of 99 patients with advanced dementia found equal mortality of about 50% over 6 months with and without feedings (70). Other studies have documented up to 90% 1-year mortality among patients who received PEGs (71). Quality of life actually may decrease in tube-fed patients due to the need for restraints or complications such as tube site pain and irritation, tube or bumper displacement, gastroesophageal reflux, or diarrhea (68,72–75).

Enteral feedings are used commonly in elderly patients with dementia, cancer, dysphagia due to stroke, and other neurologic conditions. Because there are questions about the effectiveness of enteral feedings in these situations, clinicians should review each case individually. If the patient is competent to make his or her own decisions, the risks and benefits should be presented and the patient's wishes honored. For patients who are unable to make their own decisions, an advance directive or health-care proxy should be sought. In all cases, goals and expected outcomes of therapy should be clearly defined.

End-stage illness has been associated with a decrease in oral intake that is believed to be a natural part of death. The shift to ketone metabolism that occurs with semi-starvation blunts the appetite and reduces the metabolic rate through alterations in cortisol and thyroid hormone metabolism (75). A concomitant reduction in gluconeogenesis from protein reduces the obligatory urine volume needed to excrete urea. Water produced through fat metabolism can contribute to total water needs. Thus, in end-stage illness, the body can subsist on a reduced intake of food and fluids. It has been suggested that such metabolic adaptations are one of the reasons why tube feedings have not been more effective in severely demented patients (73,76). Because such patients are not actually starving in this state, they would not be expected to benefit from supplemental feedings (76). If an acceptable BMI is being maintained with little or no weight loss, enteral feedings should not be instituted based solely on poor oral intake. Instead, it may be helpful to weigh patients monthly while offering intensive assistance with high-calorie, high-protein oral intake.

Despite the lack of evidence showing benefits of tube feedings in the geriatric patient, certain elderly patients do benefit from enteral nutrition support. When a short-term condition precludes adequate oral intake, enteral feedings can prevent a rapid decline in lean body mass and fat stores that could lead to complications associated with malnutrition. Such conditions might include certain types of cancer and stroke with prolonged dysphagia. For patients who have head and neck cancer, early placement of PEG tubes has been shown to ameliorate weight loss, reduce hospital admissions, and improve quality of life (68,77). Dysphagia occurs in 27% to 50% of patients after stroke, with up to 27% eventually regaining the ability to swallow (75). For those patients, enteral feedings can sustain life during rehabilitation until full oral feedings can be resumed.

Even elderly persons with dementia can benefit from enteral feedings if properly identified. Mueller and colleagues (78) reviewed the records of 67 elderly nursing home patients and found that those who were able to participate actively in physical therapy while receiving enteral nutrition gained weight and improved their physical function. The authors concluded that enteral feedings should be used in residents who cannot consume sufficient nutrition by mouth and who are candidates for physical therapy, as determined by a licensed physical therapist. Rimon and associates (71) examined 674 patients in a prospective observational study and found a difference in survival between subgroups of demented patients. Patients with stroke older than 80 years of age had a median survival of less than 6 months, but those younger than age 80, particularly women, had a median survival of nearly 16 months. Twenty percent of the patients in the study lived as long as 4 years after PEG placement.

Better guidelines are needed to identify those who will benefit most from enteral feedings, the optimal timing for initiation of feedings, and the best protocols to reduce complications. Limitations to the current evidence include a lack of prospective, randomized, controlled trials, differences in the definition and detection of complications such as aspiration, and heterogeneity among study participants. Both the American Society of Parenteral and Enteral Nutrition and the American Dietetic Association recommend erring on the side of feeding patients (79). Legal and ethical precedents support the patient's right to choose. When the goal of medical treatment is curative or rehabilitative, nutrition support with defined goals should be part of the plan. If the patient is acutely ill and the long-term prognosis is not evident, a time-limited trial of nasogastric feedings may be helpful.

Parenteral Nutrition

Enteral nutrition is always the preferred method of feeding when the patient is unable to ingest adequate oral nutrition and the gastrointestinal tract is functional. Indications for parenteral nutrition support include a nonfunctional gastrointestinal tract, obstruction of the gastrointestinal tract, prolonged ileus, severe gastrointestinal hemorrhage, severe diarrhea or malabsorption, mesenteric ischemia, and peritonitis. Patients given the choice

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between enteral and parenteral nutrition choose intravenous feeding (80) because of the perception that intravenous feedings will be more comfortable than tube feeding. Often, patients and families are not advised regarding the risks and complications associated with parenteral nutrition (80), such as air emboli, venous thrombosis, and sepsis. Clinicians need to educate patients and their families regarding the risk factors involved with parenteral nutrition support to allow them to make an informed decision.

Very few studies have examined the tolerance for parenteral nutrition in elderly individuals (81). Possible negative effects from the parenteral substrates could include glucose intolerance and poor lipid clearance. The ability of elderly patients to tolerate hypertonic glucose and lipid emulsions is an area for further investigation (81). Intravenous infusions of amino acid solutions in older patients have been well tolerated (82). Older adults must be monitored very carefully to ensure adequate hydration, sufficient macronutrients, and correction of metabolic anomalies to produce a positive therapeutic outcome.

Home Nutrition Support

Any elderly patient for whom home nutrition support is being considered should receive an assessment to determine whether the patient and family can handle the therapy. Impairments that may have an impact on the success of home nutrition support include alterations of vision; compromised hearing; and loss of fine motor skills, coordination, strength, and cognitive function. Successful home nutrition support depends on the ability of the patient and caregiver to manage care and obtain advice and guidance when problems arise. Another important factor is a thorough evaluation of the patient's social circumstance. The patient's financial situation, including access to private health insurance or Medicare or Medicaid, Social Security benefits, and other available sources of support, should be evaluated. The health-care professional must evaluate whether the patient and the caretaker can become independent with the treatment modalities. A recent study by Silver and associates (83) examining home enteral nutrition in the elderly revealed that most patients experienced complications that interrupted feedings and were associated with unscheduled health-care visits and hospital readmissions. These authors suggested that home enteral nutrition support requires frequent monitoring, reassessment, and interventions from a highly skilled multidisciplinary team that includes a dietitian. Potential candidates for home enteral and parenteral nutrition should not be excluded solely on the basis of age.

Conclusion

Nutrition plays multiple roles in successful aging. Added years at the end of the life cycle can be healthful, enjoyable, and productive if chronic diseases and conditions can be prevented or managed effectively. Because many chronic conditions affecting the elderly can benefit from nutrition intervention, it is incumbent upon dietitians to understand the unique nutritional needs of the elderly population and to design appropriate nutritional interventions that may include oral, enteral, or parenteral nutrition support. Dietetics professionals have an important challenge to understand the physiologic changes of the aging process, address the risks and burdens associated with nutrition support, and support ongoing research to benefit this growing population.

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References

1. Wilmoth JR. Demography of longevity: past, present and future trends. *Exp Gerontology.* 2000;35:1111–1119.
2. Mitchell-Eady C. Nutritional assessment of the elderly. In: Chernoff R, ed. *Geriatric Nutrition: The Health Professional's Handbook.* Sudbury, Mass: Jones and Bartlett Publishers; 2006:427–452.
3. Smith D, Tillipman H. *Current Population Reports.* Washington, DC: United States Census Bureau; September 2000.
4. Baker B, Wellman N. Nutrition concerns in discharge planning for older adults: a need for multidisciplinary collaboration. *J Am Diet Assoc.* 2005;105:603–607.
5. Sullivan DH. Undernutrition in older adults. *Ann Long Term Care.* 2000;8: 41–46.
6. Verdery RB. Clinical evaluation of failure to thrive in older people. *Clin Geriatr Med.* 1997;13:769–778.
7. Heymsfield SB, Tighe A, Wang Z-M. Nutritional assessment by anthropometric and biochemical methods. In: Shils ME, Olson JA, Shike M, eds. *Modern Nutrition in Health and Disease.* 8th ed. Philadelphia, Pa: Lea & Febiger; 1994.
8. Potter JF, Schafer DF, Bohi RL. In-hospital mortality as a function of body mass index: an age-dependent variable. *J Gerontol.* 1998;43:M59–M63.
9. Solomons NW. Demographic and nutritional trends among the elderly in developed and developing regions. *Eur J Clin Nutr.* 2001;23:343–350.
10. Mackle TJ, Touger-Decker R, O'Sullivan Maillet J, Holland B. Registered dietitians' use of physical assessment parameters in professional practice. *J Am Diet Assoc.* 2003;103: 1622–1623.
11. Baumgartner RN, Waters DI, Gallagher D, Morley E, Garry PJ. Predictors of skeletal muscle mass in the elderly men and women. *Mich Aging Dev.* 1999;107:123–136.
12. Corpus E, Hartmann SM, Blackman MR. Human growth hormone and human aging. *Endo Cr Rev.* 1993;14: 20–39.
13. Rudman D, Feller AG, Nagraj HS. Effects on human growth hormone in men over 60 years old. *N Engl J Med.* 1990;323:1–6.
14. Crist DM, Peake GT, Egan PA, Waters DL. Body composition response to exogenous growth hormone during training in highly conditional adults. *J Appl Physiol.* 1988;65:579–584.
15. Roe DA. *The Physiology of Aging. Geriatric Nutrition.* 3rd ed. Englecliffs, NJ: Prentice Hall; 1992:19.
16. Erlinger TP, Pollack H, Appel LJ. Nutrition related cardiovascular risk factors in older people: results from the Third National Health and Nutrition Examination Survey. *J Am Geriatr Soc.* 2000;48:1486–1489.
17. Lewis JF, Maron BJ. Cardiovascular consequences of the aging process. In: Lowenthal DT, ed. *Geriatric Cardiology. Cardiovascular Clinics.* Vol. 22. Philadelphia, Pa: FA Davis; 1992.
18. American Heart Association. *Heart Disease and Stroke Statistics – 2004 Uptake.* Dallas, Tex: American Heart Association; 2003.

19. Fair JM. Cardiovascular risk factor modification: is it effective in older adults? *J Cardiovasc Nurs.* 2003;18:161–168.
20. Ryan JJ, Zawada ET. Renal function and fluid and electrolyte balance. In: Rosenthal RA, Zenilman ME, Katlic MR, eds. *Principles and Practice of Geriatric Surgery.* New York, NY: Springer-Verlag; 2001.
21. Fuiano G, Sund S, Mazza G, et al. Renal hemodynamic response to maximal vasodilating stimulus in healthy older adults. *Kidney Int.* 2001;59:1052–1058.
22. Phillips PA, Rols BJ, Ledingham JG, et al. Reduced thirst after water deprivation in healthy elderly men. *N Engl J Med.* 1984;311:753–759.
23. Hurwitz A, Bindy DA, Schane E. Gastric acidity in older adults. *JAMA.* 1997;278:659–662.
24. Katelaris PH, Seow F, Napoli J. Effect of age, *Helicobacter pylori* infection and gastritis with atrophy on serum gastrin and gastric acid secretion in healthy men. *Gut.* 1993;34:1032–1037.
25. Firth M, Pranterk CM. Gastrointestinal motility problems in the elderly patient. *Gastroenterology.* 2002;12:1688–1700.
26. McEvoy S, Dulton J, James O. Bacterial contamination of the small intestines is an important cause of occult malabsorption in the elderly. *BMJ.* 2003;287:789–793.
27. Duman DG, Bor S, Ozutemiz O, et al. Efficacy and safety of *Saccharomyces boulardii* in prevention of antibiotic associated diarrhea due to *Helicobacter pylori* eradication. *E Eur J Gastroenterol Hepatol.* 2005;17:1357–1361.
28. Yank, Polk DB. Probiotics as functional foods in the treatment of diarrhea. *Curr Opin Clin Nutr Metab Care.* 2006;9: 717–721.
29. Madsen JL. Effects of gender, age, and body mass index on gastrointestinal transit time. *Kig Dis Sci.* 1992;37: 1548–1553.
30. Mason DL, Brunicardi FC. Hepatobiliary and pancreatic function. In: Rosenthal RA, Zenilman ME, Katlic MR, eds. Principles and *Practices of Geriatric Surgery.* New York, NY: Springer-Verlag; 2001.
31. Ratner J, Lisbona A, Rosenbloom M, Palayew M, Szabolcsi S, Tupaz T. The prevalence of gallstones disease in the very old institutionalized persons. *JAMA.* 1991;265:902–903.
32. Currie MS. Immunosenecence. *Compr Ther.* 1992;18:26–34.
33. Vargas CM, Kramarow EA, Yellowitz JA. *The Oral Health of Older Americans.* Aging Trends; No.3. Hyattsville, Md: National Center for Health Statistics; 2001.
34. Jensen GL, Friedmann JM, Coleman CD, Smicikla-Wright H. Screening for hospitalization and nutritional risks among community-dwelling older persons. *Am J Clin Nutr.* 2001;74:201–205.
35. Robertson RG, Montagnini M. Geriatric failure to thrive. *Am Fam Physician.* 2004;70:343–350.
36. Grabowski DC, Campbell CM, Ellis JE. Obesity and mortality in elderly nursing home residents. *J Gerontol.* 2005;9:1184–1189.
37. *Extending Life, Enhancing Life: A National Research Agenda on Aging.* Washington, DC: National Academy of Sciences; 1991. Available at: http://www.nap.edu/openbook/0309043999/html. Accessed July 2007.
38. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. *JAMA.* 2005;293:1861–1867.
39. Alibhai SMH, Greenwood C, Payett H. An approach to the management of unintentional weight loss in elderly people. *CMAJ* 2005;172:773–780.
40. Corrada MM, Kawas CH, Mozaffar F, Paganini-Hill A. Association of body mass index and weight change with all-cause mortality in the elderly. *Am J Epidemiol.* 2006;163:938–949.
41. Wakimoto P, Block G. Dietary intake, dietary patterns, and changes with age: an epidemiological perspective. *J Gerontol.* 2001;56A (Special Issue 11): 65–80.
42. Morley JE. Decreased food intake with aging. *J Gerontol.* 2001;56A (Special Issue 11): 81–88.
43. Chernoff R. Protein and older adults. *J Am Coll Nutr.* 2004;23:627S–630S.
44. Campbell WW, Carnell NS, Thalacker AE. Protein metabolism and requirements. In: Chernoff R, ed. *Geriatric Nutrition: The Health Professional's Handbook.* Sudbury, Mass: Jones and Bartlett Publishers; 2006:15–22.
45. Milne AC, Avenell A, Potter J. Meta-analysis: protein and energy supplementation in older people. *Ann Intern Med.* 2006;144:37–48.
46. Milne AC, Potter J, Avenell A. Protein and energy supplementation in elderly people at risk from malnutrition. *Cochrane Database Syst Rev.* 2005;1: CD003288.
47. Dunne JL, Dahl WJ. A novel solution is needed to correct low nutrient intakes in elderly long-term care residents. *Nutr Rev.* 2007;653:135–138.
48. Wilson MMG, Purushothaman R, Morley JE. Effect of liquid dietary supplements on energy intake in the elderly. *Am Soc Clin Nutr.* 2002;75: 944–947.
49. Chernoff R. Micronutrient requirements in older women. *Am J Clin Nutr.* 2005; 81(suppl):1240S–1245S.
50. Russel RM. Factors in aging that effect the bioavailability of nutrients. *J Nutr.* 2001;131:1359S–1361S.
51. Andres E, Loukili, NH, Noel E, et al. Vitamin B_{12} (cobalamin) deficiency in elderly patients. *CMAJ.* 2004;171: 251–259.
52. A Report of the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes and its Panel on Folate, Other B Vitamins, and Choline and Subcommittee on Upper Reference Levels of Nutrients, Food and Nutrition Board, Institute of Medicine. *Dietary Reference Intakes for Sub Thiamin, Riboflavin, Niacin, Vitamin B_6, Folate, Vitamin B_{12}, Pantothenic Acid, Biotin, and Choline.* Washington, DC: The National Academies Press; 1998.
53. Smith AD. Folic acid fortification: the good, the bad, and the puzzle of vitamin B-12. *Am J Clin Nutr.* 2007;85:3–5.
54. Morris MS, Jacques PF, Rosenberg IH, Selhub J. Folate and vitamin B-12 status in relation to anemia, macrocytosis, and cognitive impairment in older Americans in the age of folic acid fortification. *Am J Clin Nutr.* 2007;85:193–200.
55. Morris MC, Evans DA, Bienias JL, et al. Dietary folate and vitamin B_{12} intake and cognitive decline among community-dwelling older persons. *Arch Neurol.* 2005;62:641–645.
56. Clarke R. Vitamin B_{12}, folic acid, and the prevention of dementia. *N Engl J Med.* 2006;354:2817–2819.
57. Rampersaud GC, Kauwell GPA, Bailey LB. Folate: a key to optimizing health and reducing disease risk in the elderly. *J Am Coll Nutr,* 2003;22:1–8.
58. Weaver CM, Fleet JC. Vitamin D requirements: current and future. *Am J Clin Nutr.* 2004;80(suppl):1735S–1739S.
59. Suter P. Vitamin metabolism and requirements in the elderly: selected aspects. In: Chernoff R, ed. *Geriatric Nutrition: The Health Professional's Handbook.* Sudbury, Mass: Jones and Bartlett Publishers; 2006:31–76.
60. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Food and Nutrition Board, Institute of Medicine. *Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride.* Washington DC: National Academies Press; 1997.
61. Nieves JW. Osteoporosis: the role of micronutrients. *Am J Clin Nutr.* 2005; 81(suppl):1232S–1239S.
62. Sikon AL, Thacker HL, Carey J, Licata AA. Secondary osteoporosis: are we recognizing it? *J Women's Health.* 2006;15:1174–1183.
63. Bischoff-Ferrari HA, Dawson-Hughes B, Willett WC, et al. Effect of vitamin D on falls: a meta-analysis. *JAMA.* 2004;291:1999–2006.

(Continued on next page)

64. Lindeman RD. Mineral requirements. In: Chernoff R, ed. *Geriatric Nutrition The Health Professional's Handbook*. Sudbury, Mass: Jones and Bartlett Publishers; 2006:77–94.
65. Finucane TE, Christmas C, Travis K. Tube feeding in patients with advanced dementia. *JAMA*. 1999;282:1365–1370.
66. Gillick M. Rethinking the role of tube feeding in patients with advanced dementia. *N Engl J Med*. 2000;342:206–210.
67. Chernoff R. Tube feeding patients with dementia. *Nutr Clin Pract*. 2006;21: 142–146.
68. DeLegge MH. PEG placement: justifying the intervention. *UpToDate*. 15.1. Available at: www.uptodate.com.
69. Murphy LM. Percutaneous endoscopic gastrostomy does not prolong survival in patients with dementia. *Arch Intern Med*. 2003;163:1351–1353.
70. Meier DE, Ahronheim JC, Morris J, Baskin-Lyon S, Morrison S. Short-term mortality in hospitalized patients with advanced dementia. *Arch Intern Med*. 2001;16:594–599.
71. Rimon E, Kagansky N, Levy S. Percutaneous endoscopic gastrostomy: evidence of different prognosis in various patient subgroups. *Age and Ageing* 2005;34:353–357.
72. Shah SH. A patient with dementia and cancer: to feed via percutaneous endoscopic gastrostomy tube or not? *Pall Med*. 2006;20:711–714.
73. Cervo FA, Bryan L, Farber S. Feeding tubes in patients with advanced dementia: the decision-making process. *Geriatrics*. 2006;61:30–35.
74. Ersek M. Artificial nutrition and hydration. *J Hosp Pall Nurs*. 2003;5:221–230.
75. McMahon MM, Hurley DL, Kamath PS, Mueller PS. Medical and ethical aspects of long-term enteral tube feeding. *Mayo Cl Proc*. 2005;80:1461–1476.
76. Hoffer LJ. Tube feeding in advanced dementia: the metabolic perspective. *BMJ*. 2006;333:1214–1215.
77. Plong WM. To PEG or not to PEG. *Pract Gastroenterol*. 2005;29:16–31.
78. Mueller C, Gilbride J, Nestle M. Enteral nutrition support in elderly residents of long-term care facilities: controversies and practical solutions. *Topics Clin Nutr*. 2003;18:13–20.
79. Krenitsky J. To PEG or not to PEG – another perspective. *Pract Gastroenterol*. 2005;29:32–37.
80. Scolapio JS, Pecco MF, Tarrosa VB. Enteral versus parenteral nutrition: the patient's preference. *JPEN J Parenter Enteral Nutr*. 2002;26:248–250.
81. Chernoff, R. Nutrition support of the older adult. In: *Geriatric Nutrition: The Health Professional's Handbook*. Sudbury, Mass: Jones and Bartlett Publishers; 2006:459–472.
82. Ferry M, Leverve X, Constans T. Comparison of subcutaneous and intravenous administration of a solution of amino acids in older patients. *J Am Geriatr Soc*. 1997;45:857–860.
83. Silver HJ, Wellman NS, Arnold DJ, Livingstone A, Byers PM. Older adults receiving home enteral nutrition: enteral regimen, provider involvement, and health care outcomes. *JPEN J Parenter Enteral Nutr*. 2004;28:92–98.

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