

# Nutrition for Patients with Hepatic Failure



Joseph Krenitsky

**Malnutrition is prevalent in patients with hepatic failure; it is also an independent risk factor for morbidity and mortality in these patients. Factors that contribute to malnutrition in patients with hepatic failure include altered metabolic rate, fat malabsorption, early satiety and impaired gastric emptying, as well as frequent hospitalizations and overzealous diet therapy. Providing increased nutrition improves nitrogen balance, increases lean body mass, and some indices of hepatic function. Although restricting dietary protein is still practiced in some institutions, most patients tolerate normal, or increased, levels of protein without exacerbation of encephalopathy when adequate medical therapy is provided. The following article addresses strategies for the clinician to overcome some of the obstacles that prevent adequate nutrition delivery in this population.**

## PREVALENCE OF MALNUTRITION

**M**alnutrition is prevalent in all forms of liver disease; from 20% in compensated liver disease to more than 80% in those patients with decompensated disease (1,2). Patients with alcoholic liver disease are reported to have a greater incidence of malnutrition than those with nonalcoholic disease (3). Protein energy malnutrition has been reported in 100% of those who receive liver transplant, and malnutrition is an independent risk factor for morbidity and mortal-

ity in these patients (4). Frequently, patients with end-stage hepatic failure will present with muscle wasting, decreased fat stores, and overt cachexia. However, many more patients will have subtle changes such as fat-soluble vitamin deficiencies, anemia from iron, folate, and pyridoxine deficiency, altered cell-mediated immune function, and slow loss of muscle mass.

## ETIOLOGIES OF MALNUTRITION IN CHRONIC LIVER DISEASE

There are a number of factors that contribute to malnutrition in patients with hepatic failure (Table 1).

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Some of these factors are related to the disease process itself, such as ascites, causing fullness and early satiety. Other factors are related to frequent hospitalizations, overzealous diet therapy, and “hospital food.” In addition, there are metabolic factors such as increased metabolic rate, fat malabsorption, and impaired glycogen stores that hasten the development and expression of malnutrition in liver disease.

### DECREASED INTAKE

Inadequate food intake is one of the primary causes of malnutrition and occurs in up to two-thirds of patients with chronic liver disease. Anorexia may result from increased circulating levels of tumor necrosis factor and leptin (5). Patients with chronic liver disease also have delayed gastric emptying compared to controls (6). In those patients with ascites, early satiety and fullness are common complaints. Frequent inpatient admissions, with periods of nothing by mouth (NPO), also contribute to decreased food intake.

### ALTERED ABSORPTION

Reduced bile secretion due to cholestasis, or compromised hepatic bile synthesis may impair micelle formation, which is essential for digestion of fat by pancreatic and luminal enzymes. The fat-soluble vitamins (A, D, E, and K) are also dependent on micelle formation. Over one-third of adult patients with chronic cholestasis have vitamin A deficiency, and 20–50% of adults with primary biliary cirrhosis are deficient in vitamin D (7–9). Undiagnosed pancreatic exocrine insufficiency may be another contributing factor to altered absorption in those patients with alcoholic liver disease. Finally, patients with cirrhosis have also been reported to have an increased incidence of small bowel bacterial overgrowth. The prevalence of small bowel bacterial overgrowth in populations with cirrhosis has been documented between 35 and 60 percent of patients (10), which may further alter nutrient absorption.

### ENERGY EXPENDITURE

The resting energy expenditure of patients with chronic liver disease is variable. Those patients with acute hepatitis or advanced stages of liver failure have

**Table 1**  
**Etiologies of Malnutrition in Cirrhosis**

#### Decreased Intake

- Anorexia
- Early satiety
- Ascites
- Altered mental status/encephalopathy
- Frequent hospitalizations

#### Decreased Absorption

- Inadequate bile flow
- Bacterial overgrowth
- Pancreatic insufficiency

#### Metabolic alterations (see Table 2)

#### Iatrogenic Factors

- Overzealous dietary restrictions
- Frequent Paracentesis
- Diuresis (micronutrient losses)
- Lactulose therapy

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an increased metabolic rate. However, hypermetabolism is not a constant feature of cirrhosis. Approximately 18% of cirrhotics have been reported with hypermetabolism, and 30% with hypometabolism (11). The mean deviation between measured and predicted energy expenditure was 11%, which was less than 200 calories per day.

### ALTERED FUEL METABOLISM

Patients with hepatic failure have “accelerated starvation,” with an early recruitment of alternative fuel sources. Cirrhotic patients demonstrate significantly increased fat oxidation and gluconeogenesis with protein catabolism after an overnight fast. It would take a healthy adult approximately 72 hours of starvation to reach the same level of fat oxidation and protein catabolism as occurs in an overnight fast in a cirrhotic patient (12,13). It is believed that the diminished hepatic and muscle glycogen stores that occur with cirrhosis is a factor in this accelerated rate of starvation. Patients without

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**Table 2**  
**Metabolic Alterations in Cirrhosis**

- Increased or decreased metabolic rate
- Glucose intolerance/insulin resistance
- Rapid postprandial gluconeogenesis
- Reduced glycogen stores
- Elevated leptin
- Elevated TNF- $\alpha$
- Decreased insulin-like growth factor-1

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adequate glycogen stores utilize increased fat and muscle protein for fuel even during short-term fasting. This contributes to the loss of subcutaneous fat and muscle wasting that is the hallmark of malnutrition. Insulin resistance and decreased levels of insulin like growth factor-1 are also believed to contribute to muscle wasting in cirrhosis. See Table 2 for a list of some of the factors affecting fuel metabolism in these patients.

## NUTRITION ASSESSMENT

Conventional nutrition assessment parameters can be misleading when used for patients with hepatic failure. Unplanned weight loss is one feature that usually accompanies malnutrition. However, patients with ascites frequently have substantial weight gain prior to hospitalizations, even in the presence of significant malnutrition. It must be recognized that patients can have substantial loss of lean muscle weight, but maintain or gain net body weight due to the presence of increasing ascites.

Serum levels of albumin and prealbumin may be altered by a decrease in synthetic function reflecting the severity of hepatic impairment. These same serum proteins may be lowered as a result of an acute-phase response to a stress (GI bleed, infection, etc). Furthermore, serum proteins can be severely reduced in those with adequate nutrition intake, and can appear normal in some patients with malnutrition. Dehydration can "normalize" serum albumin, and corticosteroid administration or renal failure can "elevate" serum prealbumin to within normal limits in the malnourished patient.

Measurement of 24-hour creatinine and triceps skinfold with estimation of mid-upper arm muscle circumference, have utility for research and monitoring muscle mass changes over time. They are of limited utility in most acute care settings, and are not sensitive to short-term changes in nutrition status. A quantitative fecal fat measurement is helpful to identify those patients with steatorrhea, and the loss of fat calories that result.

Subjective assessment of muscle mass and physical signs of micronutrient deficiency upon physical exam is useful to help estimate nutrition status. Those patients with temporal muscle wasting, or obvious wasting of upper extremity muscle mass generally require attention to nutrition issues. Some patients will present with the angular stomatitis, glossitis, magenta tongue, or dermatitis of severe vitamin deficiency.

A careful review of the patient's recent oral intake is also a reasonable approach to assess nutrition risk. It is important to assess both the quality and quantity of food and beverage intake. The utility of a review of recent oral intake is dependent upon the reliability of the patient and/or family as a historian.

## Micronutrients

Patients with hepatic failure are at increased risk of deficiencies of several micronutrients. Those patients with ongoing alcohol ingestion are clearly at further increased risk of thiamine, magnesium and folate deficiency (13). Supplemental thiamine should be provided to these patients before feedings begin. Patients with compromised nutrition status are at risk for sub-optimal status of most micronutrients; a multiple vitamin is appropriate for many patients. Iron supplements or a multi-vitamin with minerals that contain iron should be avoided until iron status is established.

Patients with a history of Primary Biliary Cirrhosis (PBC), or Primary Sclerosing Cholangitis (PSC) have a significantly increased incidence of Vitamin A, D, and E deficiency (7,8). In some populations studied with PBC, the frequency of vitamin A deficiency was >33% of the population studied. Over one third of adult patients with chronic cholestasis have vitamin A deficiency, and 20–50% of adults with primary biliary cirrhosis are deficient in vitamin D. Recommended intakes of fat-soluble

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**Table 3**  
Suggested Fat-Soluble Vitamin Replacement in Cholestasis in Deficient Patients (9)

<b>Vitamin A</b>	25,000–50,000 IU per day (as Aquasol A)
<b>Vitamin D</b>	12,000 to 50,000 IU per day, and recheck in 1–2 months
<b>Vitamin E</b>	10–25 IU/kg/day

vitamins are substantially higher in those patients with cholestatic disease (Table 3). Patients with alcoholic liver disease may also have undiagnosed pancreatic exocrine insufficiency, which can lead to losses of fat-soluble vitamins. Patients with cholestatic liver disease and those with a suspicion of fat malabsorption should have levels of vitamin A, 25-hydroxy vitamin D and Vitamin E checked at baseline, and rechecked each year (7–9). Recent reports document a high incidence of vitamin A and D deficiency in patients evaluated for liver transplantation, regardless of the presence of cholestasis. However, there is a limited data regarding the accuracy of serum vitamin A in patients with decreased serum retinol binding protein levels. Unfortunately it is not clear whether serum vitamin A reflects hepatic stores in this population. Empiric supplementation of vitamin A without documentation of deficiency should be avoided. Increased vitamin A intake that is well below the toxic level has recently been demonstrated to be an independent risk for bone fractures in healthy adults (14). Patients with cirrhosis are particularly vulnerable to fractures. In a recent study, dual energy X-ray absorptiometry (DEXA) scans reflected osteopenia or osteoporosis in 55% of liver transplant candidates, irrespective of the presence of cholestasis (15).

**Zinc**

Zinc deficiency is common in patients with cirrhosis. Decreased dietary intake of meats, increased urinary excretion of zinc due to diuretic use, and increased zinc needs have been suggested as causes (16). Zinc is essential for the function of over 300 enzymes, including those of the urea cycle. Some reports have suggested that supplemental zinc may improve encephalopathy scores.

However, recent randomized trials have failed to document a significant improvement of encephalopathy scores despite a normalization of serum zinc levels (16–17).

**NUTRITION REQUIREMENTS**

**Calories**

Some patients with hepatic failure have increased calorie expenditure. However, the energy expenditure of patients with chronic liver failure is variable, and the degree of hypermetabolism, when present, is modest (11). Due to the increased prevalence of malnutrition in this population, it is often best to begin nutrition at reduced calorie levels for the first 2–3 days to decrease the severity of "Refeeding Syndrome." Refeeding syndrome can occur in patients who have adapted to starvation, and then receive increased calories (especially carbohydrate). The increased calorie provision results in increased endogenous insulin production, causing a decrease (at times dramatic) in serum potassium, magnesium, and phosphorus as these ions move from the intravascular, into the intracellular space (18). Refeeding syndrome is also associated with increased cardiac and respiratory rate, as well as fluid and sodium retention. It is recommended that patients who are at risk of "Refeeding Syndrome" receive an initial calorie provision of 15–20 calories per kg of estimated euvolemic weight. For the first 3–4 days of consistent nutrition provision, check serum potassium, magnesium, and phosphorus and replace as needed. Given the predominance of alcohol abuse in this patient population, precautionary supplemental thiamine should also be provided (18).

In patients with significant ascites, base calorie needs on an estimated "euvolemic" weight to prevent overfeeding. Estimation of euvolemic weight is subjective, and based on limited data, but is useful to avoid gross overestimation of nutrition requirements (Table 4).

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**Table 4**  
Estimated Fluid Excess in Patients with Ascites

• Mild Ascites	3–5 kg
• Moderate Ascites	7–9 kg
• Severe Ascites	14–15 kg

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**Table 5**  
**Suggested Caloric Requirements Per Kilogram**  
**of Estimated Euvolemic Weight**

Refeeding risk:	15–20 calories/kg euvolemic weight
Maintenance:	25–30 calories/kg euvolemic weight
Anabolism:	30–35 calories/kg euvolemic weight

There are a number of formulas used to estimate caloric expenditure; all have limitations in accurately predicting calorie expenditure in individual patients. Indirect calorimetry can quantify a patient’s hyper- or hypometabolism, and is essential for research purposes. However, there are no compelling data that the routine clinical use of precise measurements of calorie expenditure will affect clinical outcomes. In view of the many factors that influence the calories ingested or delivered into patients in most clinical circumstances, an estimate of calorie needs that prevents gross overfeeding or underfeeding is reasonable. Table 5 provides suggested calorie requirements based on goals of therapy.

**Protein Requirements**

Protein requirements in end stage hepatic failure remain a matter of controversy. Conventional wisdom and textbook information continue to include protein restriction to reduce hepatic encephalopathy. However, controlled trials have demonstrated that patients with hepatic failure not only require increased protein intake to maintain nitrogen balance, but commonly tolerate normal or increased protein intake without exacerbating encephalopathy. Current recommendations are to provide adequate medication (lactulose, Neomycin, Metronidazole) to control encephalopathy, and optimize protein to as much as the patient is able to tolerate (19–23). In practice, it is clear that that an *acute* worsening of encephalopathy or mental status is invariably related to infections, GI bleeding, medications, or missed doses of lactulose regardless of protein intake.

In one study, hospitalized patients with alcoholic liver disease were randomized to a regular diet, or a regular diet plus supplemental tube feedings (22). The tube feedings provided 1.5 grams of protein per kg (39 calories/kg). Not only did this protein “load” not worsen encephalopathy, but also the group who received more

protein actually had a more rapid improvement in encephalopathy scores! In another study, 136 patients with alcoholic hepatitis, were evaluated over a 28-day period (23). The authors found that a higher protein intake was associated with improved hepatic encephalopathy compared to those with a lower protein intake.

In many institutions it is still routine to place encephalopathic patients on a protein-restricted diet with an arbitrary number of protein grams per day (40 to 60 grams commonly), regardless of the patients weight, or the adequacy of their oral intake. It should first be determined if the patient is actually consuming food in significant amounts before imposing any dietary restrictions. Patients consuming significant amounts of protein who appear *resistant* to standard medical therapy might benefit from a trial of 0.8 g of protein per kg of euvolemic weight until mental status improves. When mental status is improved protein intake should be liberalized in malnourished patients to promote adequate nitrogen balance.

**Transjugular Intrahepatic Portosystemic Shunt (TIPS)**

Patients who have received a portal-caval shunt (such as TIPS) are at increased risk of encephalopathy (24) and therefore, may benefit from some type of protein restriction. One positive aspect of TIPS however, is that nutritional status is often significantly improved (6 weeks post-TIPS as measured by mid-arm muscle circumference) (25).

**Parenteral Protein**

At least one study supports the idea that parenteral protein will be less likely to precipitate encephalopathy (24). Dietary protein is converted by colonic bacteria into ammonia. Parenteral protein provision will of course not increase ammonia generation in the colon.

Suggested protein provision:

- 1.0 to 1.5 g/kg euvolemic weight as tolerated;
- 0.8 g/kg if refractory encephalopathy is present.

**Branched Chain Amino Acids (BCAA)**

Early reports of benefits of branched-chain amino acid enriched formulas to improve encephalopathy gener-

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**Table 6**  
**Comparison of Hepatic with Standard Enteral Products\***

<i>Product</i>	<i>Calories/kg</i>	<i>Protein g/1000 Kcals</i>	<i>% Fat (%MCT)</i>	<i>Cost/2000 Kcal**</i>
<b>Hepatic Products</b>				
Hepatic-Aid II (Hormel Health Labs)	1.2	35	28 (0)	\$99.50
NutriHep (Nestle)	1.5	26 High BCAA	12 (66)	\$71.00
Suplena (Ross)	2.0	15	43 (0)	\$8.59
<b>Standard Products</b>				
Nutren 1.5 (Nestle)	1.5	40	40 (50)	\$7.44
Isosource 1.5 (Novartis)	1.5	45	38 (30)	\$8.89
Jevity 1.5 (Ross)	1.5	43	30 (19)	N/A

**Manufacturer Contact Information**Hormel Health Labs: 800/533-2000 ext. 5781 or [www.hormelhealthlabs.com](http://www.hormelhealthlabs.com)Nestle: 800/776-5446 or [www.nestleclinicalnutrition.com](http://www.nestleclinicalnutrition.com)Ross: 800/544-7495 or [www.ross.com](http://www.ross.com)Novartis: 800/466-6380 or [www.novartisnutrition.com](http://www.novartisnutrition.com)

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\*\*Cost based on home delivery fees including shipping and handling for May 2003

ated enthusiasm for a dietary approach to treating encephalopathy. However, controlled trials of these products have failed to provide evidence of a significant benefit, compared to conventional medications with standard protein (26). The use of these products should be limited to those patients with intractable encephalopathy, as most patients tolerate normal protein formulas with conventional medications. Table 6 includes a comparison of several commercially available hepatic and standard enteral products.

**NUTRITION THERAPY—ORAL**

Nutrition therapy in hepatic failure should attempt to optimize oral intake and prevent or treat malnutrition. Provision of increased oral nutrition to patients with mild to moderate liver disease improved nitrogen balance and lean body mass (2).

**Avoid Prolonged Periods of NPO**

Due to inadequate glycogen stores, muscle protein is oxidized to provide blood glucose during periods of fasting (short or long). Many ESLD patients can have slow gas-

tric emptying, and ascites often prevents consumption of large meals. Frequent meals and snacks reduce the muscle breakdown between meals, and improve nitrogen balance. A bedtime snack is critical to help reduce the breakdown of lean muscle mass during the overnight fast (12). Small, frequent meals also address the early satiety that many patients experience. Oral liquid supplements have been shown to help increase calorie and protein consumption, and may allow faster gastric emptying than a solid meal. Consider adding D<sub>5</sub> to intravenous fluids if NPO status is required for procedures, etc.

**Avoid Unnecessary Diet Restriction**

A low sodium diet is helpful in the management of ascites, but in patients with minimal intake, it may be helpful to avoid ANY dietary restrictions. Many patients do not require a protein restriction when adequate medical therapy for encephalopathy is administered. Reassess need for any diet modification as intake improves. One frequently overlooked issue is that many patients do not have adequate dentition to consume a regular diet. Modification of diet to meet ability to chew may improve oral intake.

### Vegetable Protein

Some studies have suggested that vegetable protein sources may offer advantages for patients with ESLD (27,28). Trials conducted to date have been small, unblinded, and have not demonstrated significant improvements in clinically apparent encephalopathy. In addition, vegetarian diets, with increased fiber content and decreased calorie and protein density of foods, require larger volumes of food intake to meet calorie and protein demands. This can be a distinct *disadvantage* in patients with early satiety or delayed gastric emptying. For a summary of recommended guidelines for enhancing oral intake see Table 7.

## NUTRITION SUPPORT

### Enteral Nutrition

Patients who are unable to meet nutrient needs despite efforts to optimize oral nutrition intake should be considered candidates for enteral tube feeding. Several

randomized trials have provided evidence that enteral feeding is more effective than diet alone, is well tolerated, and can lead to improvement of some indicators of hepatic function (2,22). Another positive aspect of feeding tube placement is that it allows for adequate and consistent lactulose therapy from above. While there may be nutritional benefits of providing enteral tube feedings, there are often a number of “barriers” that prevent the full utilization of tube feedings in patients with hepatic failure (Table 8).

In general, it may be beneficial to choose nutrient-dense formulas (1.5 to 2.0 calorie/mL). The use of calorie dense enteral products allows for provision of full calorie needs in a smaller volume and lower hourly rate. This can be an advantage in those patients who have delayed gastric emptying, complain of “fullness,” or who require a fluid restriction. Patients with steatorrhea or impaired bile flow may benefit from a formula with a portion of the lipid content as medium chain triglyceride (MCT). Controlled trials have demonstrated that most patients tolerate the protein in standard tube feeding formulas without precipitation of encephalopathy (22).

Trials of specialized branched chain amino acid (BCAA) formulas are limited by the small number of patients enrolled, the lack of adequate feeding in the “control” group, or the failure to provide standard “anti-encephalopathic” agents (such as lactulose) during the study (26). Branched chain amino acid formulas should be reserved for those patients who appear intolerant of adequate protein from standard formulas. See Table 6 for a comparison of hepatic and standard formulas available.

### Esophageal Varices/GI Bleeding

Placement of a small bore, polyurethane (soft, “dobhoff-type”) nasoenteric feeding tube does not appear to cause a greater incidence of rebleeding from varices, compared to no feeding tube (29). Patients who are malnourished or NPO greater than 5 days, who have a stable hematocrit and do not have an imminent (next 24 hours) endoscopic procedure planned, should be considered for placement of a feeding tube.

**Table 7**  
**Suggested Guidelines for Improving Oral Intake \***

- Avoid prolonged periods of NPO
- Provide small meals and snacks during the day
- Encourage an evening snack to reduce duration of overnight fasting
- Encourage oral liquid supplements
- Avoid any unnecessary diet restrictions
  - Avoid protein restriction, except in TIPS and refractory encephalopathy
  - Avoid any dietary restrictions during periods of extremely poor intake
  - Tailor sodium restriction to absolute need
- Provide foods appropriate for chewing/swallowing abilities
- Optimize gastric emptying
  - Avoid excessive fiber
  - Control blood glucose
  - Avoid gut-slowing drugs where possible
  - Use liquids over solids if necessary

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**Table 8**  
**Barriers to Enteral Nutrition Support in Hepatic Failure\***

<i>Barriers</i>	<i>Options</i>
Patient reluctant to have nasogastric tube	Emphasize the positive aspects of good nutrition as an essential part of their care
Nasogastric placement as “a threat” to induce oral intake	Encourage nutrition support as supportive therapy if necessary
Concern for variceal bleeding	Use of soft, polyurethane tubes
Frequent dislodgement of nasogastric tube	Consider nasal bridle, hand mitts, or a “sitter” with the patient
Diarrhea	Avoid excessive lactulose, sorbitol-containing medications, etc., check for <i>Clostridium Difficile</i>
Nausea	Use a nutrient-dense feeding, antiemetics, consider jejunal placement of feeding tube
Inability to place PEG in patient with ascites	Use nasogastric for short-term feedings; optimize oral intake when possible

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**Mental Status/Tube Displacement**

Patients who have compromised mental status, may not be cooperative with nasogastric tube placement, and are at risk for frequent self-removal of the feeding tube. In select patients a “nasal bridle” can be an acceptable method to discourage self-removal of feeding tubes (30). Although to some this may seem an extreme measure, frequent reinsertions of feeding tubes is not without risk or discomfort.

**Patient Acceptance**

Occasionally, placement of a nasogastric tube is presented as a form of “punishment” (“if you don’t start eating, we will have to place a feeding tube...”) that will result if the patient is unable, for whatever reason, to eat. Some patients equate nasogastric tubes with a prior experience with a large-bore (Salem sump-type) tube used for suction. If this is the case, showing the patient a small bore feeding tube may allay their fears. Furthermore, if patients are informed of the benefits of enteral feeding, they are more likely to accept nasogastric tube placement.

**Long-Term Access**

Placement of a PEG or J-tube is generally contraindi-

cated in patients with significant ascites. In select patients it may be desirable to maintain nasogastric tube placement after discharge for several weeks, to allow time for improved nutrition status.

**Parenteral Nutrition**

Parenteral nutrition is associated with increased infectious and metabolic complications compared to enteral feeding. Parenteral nutrition should be reserved only for those patients who cannot receive enteral nutrients, (ileus, small bowel obstruction, etc.). Peripheral parenteral nutrition has limited utility, due to the increased fluid volume required to provide significant calories and protein. While there is evidence that parenteral protein will be less likely to precipitate encephalopathy (24), the presence or fear of encephalopathy is generally not sufficient reason to begin TPN in a patient with a functioning GI tract. As discussed earlier, the vast majority of patients with ESLD will tolerate standard tube feeding formulas without exacerbating encephalopathy.

The optimal composition of macronutrients in TPN for patients with hepatic failure is unclear. TPN that provides either excessive lipid or dextrose has been

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implicated in exacerbating hepatic compromise. Until data from prospective studies are available, when parenteral nutrition is required, this author recommends limiting IV lipid provision to less than one gram of long chain fat per kg and providing a balanced fuel source.

### CONCLUSIONS

Hepatic failure is associated with a high prevalence of malnutrition. Malnutrition, in turn, is associated with compromised outcomes in this population. The cause of malnutrition in patients with cirrhosis is multifactorial, and includes decreased intake, altered metabolism, and increased nutrient losses. Oral intake can often be enhanced with careful attention to individual needs and by avoiding unnecessary diet restrictions. Enteral feedings are a safe and effective way to provide increased nutrition when a patient is unable to meet their basic needs by mouth. Standard feeding formulas are well tolerated by most patients, and further data would be valuable to understand the role of specific nutrients on the outcomes of patients with hepatic failure. ■

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