Although gastric resections are performed less frequently today, clinicians are still faced with treating patients who have a history of gastric surgery. Nutritional intolerances and malabsorption can lead to nutrient deficiencies and undesirable clinical consequences. Intolerances can often be managed with dietary manipulation and close nutrition follow-up. Nutrient deficiencies leading to anemia and metabolic bone disease require ongoing monitoring and supplementation. This article describes the various gastric resections and provides guidelines for the management of both acute and long-term nutrition-related side effects.

INTRODUCTION

Today, gastric resection is reserved for patients with peptic ulcer disease that has failed to respond to medical therapy or those with malignant disease. Steadily declining cancer rates and improved medical therapy for ulcer disease has fortunately reduced the need for this type of surgery. However, clinicians often treat patients with a history of gastric resection.

Gastric resections can be divided into two categories: partial or subtotal gastrectomy (PG) and total gastrectomy (TG). Similar nutritional complications may result from either surgery. Timely and appropriate nutritional intervention can minimize diet intolerances, weight loss and micronutrient deficiencies that often follow. This article will review the various types of gastric resections and provide guidelines to help health care professionals manage and prevent both acute and long-term nutrition-related side effects.

GASTRIC RESECTIONS

Partial Gastric Resection

A PG may be used in the treatment of ulcers that are resistant to standard therapy, ulcers that continue to recur despite aggressive treatment or ulcers that cause
severe complications (1). A PG is also used as treatment for gastric malignancies restricted to the antrum (2). PG involves removal of the gastrin-secreting antrum (up to 75% of the distal stomach) (1). Reconstruction is performed with anastomosis of the remaining gastric segment to the duodenum, a Billroth I (BI), or to the side of the jejunum (approximately 15 centimeters distal to the ligament of treitz), a Billroth II (BII) (1) (see Figures 1–4). The duodenal stump is preserved in the Billroth II to allow continued flow of bile salts and pancreatic enzymes (3). However, because of dysynchrony of food and bile/enzyme entry, patients with a BII may still have inadequate mixing (4). Today, BI operations are rare and are used primarily for very small tumors in the antrum (5).

Vagotomy
BI and BII operations may or may not involve vagotomy. Furthermore, the type of vagotomy may differ. A truncal vagotomy severs the vagus on the distal esophagus. It significantly reduces acid secretion and creates gastric stasis and poor gastric emptying and is therefore combined with a drainage procedure (pyloroplasty or gastrojejunostomy) (1). A selective vagotomy divides and severs the vagus nerve branches that supply the parietal cells while preserving those that innervate the antrum and pylorus. Thus, a drainage procedure is unnecessary, and the innervation to other organs is preserved (1). Unfortunately, a selective

(continued on page 66)
vagotomy is more technically difficult and is associated with a higher rate of ulcer recurrence (1). A subtotal gastrectomy as treatment for cancer is similar to a Bill but denervates the vagus only on the resected area of the stomach. A detailed operative note is important to determine the procedure performed.

Roux-en-Y Total Gastric Resection

TG’s are performed for gastric malignancies that affect the middle or upper part of the stomach. The entire stomach is resected and standard reconstruction is usually via the Roux-en-Y method (6). Doubling the end of the Roux limb and performing a side-to-side anastomosis is sometimes used to create a “stomach pouch.” The Roux limb is of sufficient length so that the esophageal anastomosis will be at least 40 centimeters above the subsequent jejunojejunostomy (6). Figures 5 and 6 illustrate a Roux-en-Y procedure without creation of a pouch. TG, by nature, involves a functional vagotomy, removing cholinergic drive and eliminating acid production.

NUTRITIONAL PRESENTATION

Studies investigating weight loss after gastric resection have found no significant difference between TG and PG patients (7,8). In addition, similar post-prandial complaints (early satiety, epigastric fullness and symptoms of dumping syndrome) have been described in both groups (7). It is clear that weight loss usually follows gastric resection with reported loss ranging from 10%–30% of preoperative weight (4,7,9,10–13). This loss has been attributed to inadequate oral intake, malabsorption, rapid intestinal transit time and bacterial overgrowth (4,9,10,11,14). More likely, it is a combination of all these factors. Nevertheless, weight gain after surgery is possible (15,16). Frequent nutrition follow-up in the early postoperative period is the key to preventing a decline in nutritional status. Indeed, several reports confirm that in the absence of nutrition follow-up, patients become progressively malnourished (15,17–19). Too often, gastrectomized patients are discharged without adequate instruction on what and how much to eat. It is therefore essential for clinicians to provide nutrition intervention and follow-up until patients demonstrate the ability to maintain or gain weight, as the case necessitates.

POST GASTRECTOMY SYNDROME

Post Gastrectomy Syndrome encompasses nutritional intolerances and deficiencies. Frequent intolerances include dumping syndrome, fat malabsorption, gastric stasis and lactose intolerance. Combinations of these are most likely responsible for acute post-operative weight loss, the most frequent complication of gastrectomized patients. Nutrient deficiencies develop
months to years after gastric resections and can result in deleterious clinical consequences. Anemia and bone disease are the most common manifestations of the nutrient deficits seen in these patients.

**NUTRITION INTOLERANCE**

**Dumping Syndrome**

Early dumping syndrome (DS) occurs about 15–30 minutes after ingesting a meal and is evidenced by diarrhea, fullness, abdominal cramps and vomiting (1). Post-prandial weakness, flushing, dizziness and sweating may also accompany early DS (1). The symptoms of DS are attributed to loss of the gastric reservoir and accelerated gastric emptying of hyperosmolar contents into the proximal small bowel (20,21). Late DS presents two to three hours after eating and results in weakness, sweating, nausea, hunger and anxiety. Late dumping is thought to be the result of reactive hypoglycemia (1).

Foods and liquids with high sugar content may exacerbate symptoms of both early and late DS.

The percentage of patients who develop dumping syndrome after a PG or TG reportedly varies from 1%–75% (4,10,12,13). Symptoms of DS are more prevalent in the immediate post-operative period and often subside over time (13). One study followed patients five years post-operatively and demonstrated reduced adverse gastrointestinal symptoms over time (11). Only about 1% of patients will develop persistent, debilitating symptoms of DS (22).

Diarrhea associated with dumping syndrome is thought to be precipitated by a high fluid intake at mealtime. One study looking at patients undergoing vagotomy found an increase in diarrhea after fluid meals and a significant decrease in intestinal motility in response to dry meals (23). Guidelines for an anti-dumping diet can be found in Table 1. DS unresponsive to diet manipulation may require meeting with a nutritionist and use of gut-slowing medication (13).

**Fat Malabsorption**

Studies looking at fat malabsorption after PG and TG have demonstrated abnormal fecal fat excretion (4,9,12,24). Jae-Moon Bae, et al found a statistically significant increase in fecal fat excretion in TG patients when compared with healthy controls (9). Of note, addition of exogenous pancreatic enzymes reduced fecal fat excretions in two study participants with severe diarrhea. One study measuring exocrine pancreatic function in TG patients found that all patients had severe exocrine pancreatic insufficiency three months after surgery (24). Tovey, et al found that 20% of patients following PG had severe steatorrhea (≥12 grams fat in stool/day) (12). Grant, et al indicates that 25% of patients after a BI demonstrate steatorrhea however, only 10% of these cases were of clinical significance. The same review states that 50% of patients with a BII have increased fecal fat, only 20% of which are clinically significant.

**Table 1**

**Anti-Dumping Diet**

- Eat 6 or more small meals a day
- Eat slowly and chew all foods thoroughly
- Sit upright while eating
- If you experience nausea, vomiting or diarrhea when consuming high-sugar foods, avoid or limit the following: Kool-aid, Juice, Soda, Ensure, Boost, cakes, pies, candy, doughnuts, cookies, fruits cooked or canned with sugar, honey, jams, jellies
- Limit fluid consumption at meals. Drink liquids 30–60 minutes either before or after meals
- Eat a protein containing food with each meal. High protein foods include the following:
  - Eggs, meat, poultry, fish, lunch meat, nuts, milk, yogurt, cottage cheese, cheese, peanut butter, dried beans, lentils, tofu
- Choose high-fiber foods when possible. These include:
  - Whole wheat bread, whole wheat pasta, fresh fruits and vegetables, beans (black, brown, pinto, kidney, garbanzo), fiber-fortified cereal

If you have difficulty maintaining your weight, you may need to drink a nutritional supplement for extra calories. You can try low-sugar over-the-counter supplements. These include no-sugar added Carnation Instant Breakfast, sugar-free Nutrishakes or Glucerna weight loss shakes.

Reprinted with permission from University of Virginia’s Digestive Health Center of Excellence. www.healthsystem.virginia.edu/internet/digestive-health/antidump.cfm
Table 2
Guidelines for Pancreatic Enzyme Supplementation

- Take capsules or tablets with meals or snacks. Typical dose is 2-3 capsules with meals and 1-2 capsules with snacks (lipase units vary per brand). Titrate dose as needed based on clinical response such as continued diarrhea or weight loss.
- To protect enteric coating, do not crush or chew the microspheres or microtablets. If swallowing of the capsules is difficult, open and shake contents into a small quantity of soft non-hot food (applesauce, jello) and swallow immediately. Viokase powder may be another alternative to opening capsules.
- Viokase Powder (Axcan Scandipharm) may be used with tube feeding. Administer 1/2 tsp/can tube feeding.

*Adapted from www.effectus.com Drug Facts and Comparisons

decreased enzyme production reduces the ratio of enzymes to food (8,24). Finally, due to loss of the antrum, and hence its sieving function, larger than normal food particles empty into the jejunum, making enzyme attack more difficult (14).

Qualitative or quantitative fecal fat may be useful in the diagnosis of fat maldigestion. For these tests to be accurate, clinicians must ensure patients consume at least 100 grams fat/day. Enzyme replacement may be necessary in those patients with clinically significant fat maldigestion. Prolonged steatorrhea may necessitate monitoring and replacement of fat-soluble vitamins (13). See Table 2 for guidelines on enzyme replacement therapy. The use of a low-fat diet with the addition of medium-chain triglycerides (MCT) to treat steatorrhea has been suggested (4). Palatability and cost make MCT a less desirable option. Refer to the May 2003 and May 2004 issues of Practical Gastroenterology for more information on the use of MCT oil (25,26).

Gastric Stasis

Three to five percent of patients with truncal vagotomy are reported to experience problems with gastric stasis (14). Use of gastroscopy is essential to distinguish patients with mechanical obstruction from those with gastric atony (1). Symptoms of poor emptying may manifest as post-prandial bloating, discomfort or fullness lasting many hours. Emesis of undigested food ingested hours to days before may also be present (14). These patients are at a higher risk for bezoar formation, bacterial overgrowth and intolerance to solid food; liquids may be processed normally or rapidly (14). Diet manipulation and/or prokinetic drugs are variably effective (1,14). For more information on gastroparesis, bezoars and bacterial overgrowth see the March 2003, January 2004 and July 2003 issues of Practical Gastroenterology respectively.

Lactose Intolerance

Lactase, the enzyme required for lactose absorption, is found primarily on villi in the jejunum (27). Most gastrectomized patients have an intact jejunum, therefore lactose intolerance, in these patients, is deemed “functional.” Patients complaining of abdominal cramping or pain, bloating, diarrhea, flatulence and distention after consumption of lactose may do well to decrease or avoid it. Tolerance to lactose is typically dose-dependent and may improve over time (27). Many patients may be able to tolerate smaller amounts of lactose containing foods throughout the day (27). Lactase enzymes are available for patients who wish to continue consuming dairy products. A thorough review of lactose intolerance may be found in the February 2003 issue of Practical Gastroenterology (27).

Although diet therapy may be beneficial in treating nutritional intolerances, it is important to minimize diet restrictions. Superfluous restrictions may cause frustration to the patient and can further aggravate weight loss. Emphasize to patients that intolerances are often short-lived. If weight loss continues despite dietary management, enteral feedings for supplemental nutrition support should be initiated. In situations where gastric remnant size precludes a gastrostomy tube, a surgical or endoscopically placed jejunostomy tube may be considered.

NUTRIENT DEFICIENCIES

Anemia

Nutritional anemias resulting from a vitamin B₁₂, folate or iron deficiency are common in gastrectomized patients. Consequences of anemia can be severe, therefore baseline and periodic monitoring are (continued on page 70)
important. Anemia often presents as a late complication of gastric resection, placing patients with a distant history of the surgery at an even greater risk.

Megaloblastic and Pernicious Anemia

Megaloblastic anemia may be the result of either vitamin B₁₂ or folate deficiency. Either vitamin will clear the anemia but folate supplementation alone can provide a deceptive cure, leaving a serious B₁₂ deficiency untreated. B₁₂ deficiency may result in PG and TG patients for numerous reasons. Normally, intrinsic factor is complexed to B₁₂ and facilitates its absorption by the terminal ileum. Reduction in intrinsic factor and reduced gastric acidity in gastrectomized patients impairs cleavage of protein bound B₁₂ (1). Bacterial overgrowth and reduced intake of B₁₂ rich foods may also contribute to a deficiency (1).

A wide range of B₁₂ deficiency has been reported in PG (10%–43%) and TG (theoretically 100%, except for supplementation with parenteral vitamin B₁₂) (8). B₁₂ deficiency has been found as early as one year post-operatively and is more common in late post-operative states (12). Lassitude, fatigability, chills, numbness in extremities, dizziness and neurological symptoms may be symptoms of B₁₂ deficiency (30). Clinical features are useful in the diagnosis of megaloblastic anemia but can be non-specific or absent in some patients (28). Therefore, periodic serum monitoring and supplementation of B₁₂ is warranted.

A recent study investigated the effects on TG patients supplemented with either oral or intramuscular supplementation (30). Interestingly, enteral B₁₂ treatment increased serum concentration rapidly. Symptom resolution was comparable in patients who received enteral and parenteral supplementation. It is possible that the body adapts after TG and may produce intrinsic factor in the duodenum and ileum (1). The decision to supplement B₁₂ orally or via intramuscular (IM) injection should be based on expected patient compliance. Tovey et al found intramuscular injections of 1000 micrograms in alternate months to be effective (12). Evidence from a 1997 investigation suggests that intranasal B₁₂, although expensive, might be an alternative mode of administration (32). For a cost comparison of oral, IM and nasal B₁₂ see Table 3. Table 4 outlines guidelines for the monitoring and supplementation of B₁₂.

Folate

Folate deficiency may develop after gastric surgery but is not well studied (14). Causes of folate deficiency are likely multifactorial including malabsorption (the first site of absorption is the duodenum) and impaired digestion (14). Red blood cell (RBC) folate should be used when diagnosing a folate deficiency. RBC folate is a better indicator of body folate stores than serum folate, which is affected by recent folate intake (28). A daily dose of 5 mg folate is recommended in defi-
Table 5
Guidelines for Iron Replacement in Adults

• One hundred fifty to 300 mg elemental iron/day in three divided doses. In general, about 4-6 months of oral iron therapy is needed to reverse uncomplicated iron deficiency anemia.
• Sustained release or enteric-coated preparations reduce amount of available iron as iron is delivered past duodenum in both BII and TG.
• Do not crush or chew sustained release preparations.
• Absorption is enhanced when iron is taken on an empty stomach but GI intolerance may necessitate administration with food.
• GI discomfort may be minimized with slow increase to goal dosage; decrease dose to 1/4–1/2 BID-QID if necessary; some is always better than none.
• Do not take within 2 hours of tetracyclines or fluoroquinolones.
• Drink liquid iron via straw to minimize dental enamel stains.

*Adapted from www.efactsweb.com Drug Facts and Comparisons

Table 6
Percent Elemental Iron in Various Iron Formulations

<table>
<thead>
<tr>
<th>Iron Source</th>
<th>% Elemental Iron Content</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ferrous Sulfate</td>
<td>20</td>
</tr>
<tr>
<td>Ferrous Sulfate, exsiccated</td>
<td>30</td>
</tr>
<tr>
<td>Ferrous Gluconate</td>
<td>12</td>
</tr>
<tr>
<td>Ferrous Fumarate</td>
<td>33</td>
</tr>
</tbody>
</table>

*Adapted from www.efactsweb.com Drug Facts and Comparisons

Microcytic Anemia

Iron deficiency is the most common anemia following gastric resection (14). The reported incidence varies tremendously. In 11 studies reviewed by Fisher, 5%-62% of patients with BII were found to be iron-deficient (33). Indeed, at 10 years post gastrectomy, iron deficiency was noted to be the most frequent nutrient deficiency (12). Iron deficiency may manifest more quickly in BII procedures, as compared with BI operations, presumably due to lack of duodenal continuity with BII (34). However, Tovey, et al found no difference in microcytic anemia incidence between BI and BII patients (12).

Alterations in digestion and absorption are thought to be responsible for iron deficiency in TG and PG patients. The duodenum, the primary site for iron absorption, is bypassed (except with BI) and reduced gastric acidity impairs the conversion of ferric iron to the more absorbable ferrous form (14). Reduced iron intake may also play a role.

Ferritin levels in the non-acute phase setting are an accurate indicator of iron stores over time (28). Iron supplementation, in the form of oral therapy, is effective in deficiency states (13). Oral iron may be given as oral ferrous sulphate, gluconate or fumarate. Optimal response occurs with approximately 200 mg elemental iron daily (28). Doses are typically administered three times daily, preferably six hours apart. The addition of vitamin C will enhance iron absorption. Of

Table 7
Elemental Iron Content of Various Iron Formulations

<table>
<thead>
<tr>
<th>Product (over-the-counter)</th>
<th>Dose</th>
<th>Elemental Iron Content (mg)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tablets</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ferrous Sulfate</td>
<td>325 mg</td>
<td>65</td>
<td></td>
</tr>
<tr>
<td>Ferrous Gluconate</td>
<td>325 mg</td>
<td>36</td>
<td></td>
</tr>
<tr>
<td>Ferrous Fumarate</td>
<td>325 mg</td>
<td>106</td>
<td></td>
</tr>
<tr>
<td>Suspension</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ferrous Sulfate Elixir</td>
<td>220mg/5ml</td>
<td>44mg/5ml</td>
<td>Brands vary may contain sorbitol</td>
</tr>
<tr>
<td>Ferrous Sulfate Drops</td>
<td>75mg/0.6ml</td>
<td>15mg/0.6ml</td>
<td>Brands vary may contain sorbitol</td>
</tr>
<tr>
<td>Feostat (ferrous Fumarate)</td>
<td>100mg/5ml</td>
<td>33mg/5ml</td>
<td>Butterscotch flavor</td>
</tr>
<tr>
<td>Chewable tablets</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feostat (ferrous Fumarate)</td>
<td>100 mg</td>
<td>33</td>
<td>Chocolate flavor</td>
</tr>
</tbody>
</table>

*Adapted from www.efactsweb.com Drug Facts and Comparisons
Table 8

Increasing Your Iron Intake
If you need to increase the iron in your diet, try these foods:

**Best Sources:**
Pork loin  |  Sardines  |  Molasses  
Oysters   |  Clams     |  Raisin Bran

**Good Sources:**
Lean Beef  |  Kidney Beans  |  Spinach  |  Enriched Macaroni
Shrimp    |  Pinto Bean   |  Greens   |  Fortified Cereals
Tuna      |  Navy Bean    |  Avocado  |  Dried Apricots
Tempeh (soy product) | Lentils |  Raisins  |  Potatoes with skin
Green Peas|  Lima Beans   |  Prunes, Figs |

**Fair Sources:**
Turkey    |  Salmon      |  Nuts     |  Strawberry
Broccoli  |  Chicken     |  Haddock  |  Peanut Butter
Banana    |  Blueberries |  Tofu     |  Cod
Tomatoes  |  Raspberries |

Try to eat foods high in Vitamin C with your iron-containing foods. (Vitamin C helps your body absorb iron from food). A serving as small as 3 oz. of orange juice or any vitamin C containing beverage will do the trick.

**Foods High in Vitamin C:**
Oranges |  Pineapple  |  Broccoli  |  Asparagus
Grapefruit |  Strawberries |  Cauliflower |  Potatoes
Lemon   |  Raspberries |  Spinach   |  Sweet Potatoes
Cantaloupe |  Tomatoes   |  Kale      |

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note, solubilization of iron tablets may not be adequate in gastrectomized patients (35). Chewable or liquid iron will ensure dissolution.

Gastrointestinal (GI) side effects such as nausea, abdominal pain, constipation or diarrhea often decrease patient compliance to iron therapy. Advising patients to take iron with food can reduce GI intolerance. Because the body increases its avidity for iron uptake in deficient states (up to 20%-30%), it is important to emphasize some iron is better than none (28). Reduced doses of one-half to one tablet once or twice daily may prevent patients from discontinuing supplementation altogether. Encouraging increased intake of iron-rich foods is also important. Emphasis should be placed on heme iron sources as they are more readily absorbed. Parenteral iron should be reserved for extreme cases due to risk of anaphylactic shock and expense. See Tables 5–8 for information on iron supplementation.

**Metabolic Bone Disease**
Bone disease, as osteoporosis, osteopenia and osteomalacia, is commonly reported in gastrectomy patients (4,10,14,36–39). Reducing fracture rates is the primary clinical goal when treating bone disease. It is therefore imperative to identify high-risk patients early and initiate therapies intended to reduce fracture incidence.

One study found that 18% of PG patients had osteomalacia based on rigorous histomorphometric diagnostic criteria (39). Of note, the majority of these patients had normal serum calcium, alkaline phosphatase and 25-hydroxyvitamin D (25-OHD). A low bone mineral density (BMD) has been reported in 27%-44% of gastrectomized patients (40). It has been postulated that older studies relying solely on lab values have underestimated the prevalence of osteomalacia (38). Klein, et al found that vertebral body fractures were three times as common in men who had undergone a BII when compared with controls (37). It is important to note that age and bone status at the time of surgery will play a role in overall bone disease independent of gastric resection.

The etiology of bone disease in gastrectomized patients is uncertain but appears to be a combination of decreased intake of calcium, vitamin D and lactose-containing foods, coupled with altered absorption and metabolism (14,37,38). One study demonstrated an increase in 25-OHD when TG and PG patients were supplemented with 400 IU of vitamin D, in the form of a multivitamin tablet, daily (10). Another study found statistically significant increases in 25-OHD in PG patients supplemented with 400–600 IU of vitamin D2 (39).

(continued on page 74)
Table 9
Summary of Nutrition Management Guidelines Following Gastric Resection

Maintain optimal nutritional status
- Determine cause(s) of weight loss through careful diet history
- Provide diet education to minimize symptoms of dumping syndrome and lactose intolerance, if present
- Daily multivitamin with minerals
- Additional calcium and vitamin D supplementation as warranted
- Continued nutrition intervention for at-risk patients

Treat fat malabsorption
- Determine if steatorrhea present (ensure patient consuming ≥100 grams fat/day when checking qualitative or quantitative fecal fat)
- Consider use of pancreatic enzymes
- Use gut-slowing agents if needed
- Treat bacterial overgrowth if present
- Monitor and supplement fat soluble vitamins as needed
- Daily multivitamin with minerals

Prevent Nutritional Anemias
- Monitor
  - Vitamin B12
  - RBC folate
  - Ferritin
- Supplement as needed (see Tables 3–8)

Prevent and treat metabolic bone disease
- Monitor 25-OHD Vitamin D
  - 1,25-dihydroxyvitamin D is not a good indicator of vitamin D status
- Supplement with Calcium and Vitamin D
  - 500 mg calcium TID
  - 800 IU vitamin D daily
- Monitor Bone Mineral Density (DEXA)
- Evaluate need for anti-resorptive and bone formation agents

Gastric Stasis
- Treat bezoars (see Practical Gastroenterology (PG) article, January 2004)
- Treat bacterial overgrowth (see PG article, July 2003)
- Treat gastroparesis (see PG article, March 2003)

However, there is not a clear relationship between BMD and 25-OHD (38). Alhava, et al demonstrated beneficial effects on BMD in men with a combination of two grams calcium and 1000 IU calciferol (41) but a follow-up study failed to show effectiveness with the same regimen (42). A recent meta-analysis suggests that vitamin D supplementation reduces the risk of falls in older individuals by more than 20% (43).

Currently, there are no accepted supplementation guidelines for calcium and vitamin D in post-gastrectomy states. Daily multivitamin tablets contain, on average, 250 mg calcium and 400 IU vitamin D, therefore additional supplementation is needed. For patients with bone disease, 1500 mg calcium and 800 IU vitamin D daily is recommended. For maximum absorption, calcium should be administered in single doses no greater than 500 mg. Patients should be encouraged to include calcium rich foods in their diet as tolerated. Refer to the February 2003 issue of Practical Gastroenterology for a list of calcium-rich foods.

Dual energy x-ray absorptiometry (DEXA) provides an inexpensive, reproducible method to determine BMD (44). Given the frequency with which bone disease affects gastrectomized patients, it is reasonable to monitor BMD, even in the setting of normal laboratory values, at baseline and then every one to two years. Prompt initiation of anti-resorptive agents (calcium, vitamin D, calcitriol and bisphosphonates) and bone-formation agents (recombinant hormone PTH) may need to be considered in severe cases.

CONCLUSION
It is clear that nutrition intervention plays an important role in patients who have undergone gastric resection. Continuous nutrition assessment and intervention is an effective tool to prevent or minimize dietary intolerances and manifestations of nutrient deficiencies. Table 9 provides a summary of nutrition management guidelines following gastric resection.

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