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# Gastroparesis: Diagnosis and Management

Project ID: 12-0013

## Learning Objectives

Upon completion of this activity, the participant will be better prepared to:

- Define gastroparesis and identify its prevalence and incidence
- Discuss the diagnosis of gastroparesis, including risk factors, symptoms, and diagnostic modalities
- Describe the contemporary management of gastroparesis, with an emphasis on balancing the risks and benefits of therapy

## Credit Designation

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## Introduction

Gastroparesis is a chronic condition that is characterized by delayed emptying of solids and liquids from the stomach in the absence of overt mechanical lesions that could account for the findings. Although there is no widely accepted definition of gastroparesis, it often presents with upper gastrointestinal symptoms, most commonly nausea, vomiting, abdominal pain, early satiety, and bloating.[1] The true prevalence of gastroparesis is difficult to estimate because of nonspecific symptoms and the lack of straightforward diagnosis; however, one large population-based study found that the age-adjusted prevalence of definite gastroparesis (defined in this study as delayed gastric emptying and typical symptoms for ≥3 months) was 24.4 per 100,000 persons.[2] In patients with diabetes, the prevalence of overt gastroparesis is as high as 18%,[3] although it has been estimated that up to 50% of patients with diabetes have some degree of delayed gastric emptying.[4]

While it is difficult to estimate the prevalence, incidence, and costs of gastroparesis, data suggest that the number of gastroparesis-related hospitalizations are increasing in the United States.[5] In fact, one study estimated that the number of hospitalizations with gastroparesis as the primary diagnosis increased by 3977 to 10,252 (158%) between 1995 and 2004 and the number of hospitalizations with gastroparesis as a secondary diagnosis increased from 46,726 to 134,146 (136%) over the same time period.[5] In the same study, gastroparesis was associated with a significantly longer hospital length of stay than gastroesophageal reflux disease, gastric ulcer, gastritis, or nonspecific nausea/vomiting, and with the second-highest hospital charges. The underlying reason for this increase is unknown, however, some combination of wider recognition of the disease, higher prevalence of diabetes, and longer survival of diabetes patients may account for some or all of the change.[5]

Gastroparesis is associated with significant morbidity, including debilitating vomiting and abdominal pain and weight loss. Gastroparesis is also associated with an inherent risk of malnutrition, poor absorption of oral medications, and—in patients with diabetes—disordered glycemic control, at least in part owing to poor absorption of both nutrients and oral antidiabetic medications. In addition, gastroparesis is associated with significantly reduced survival compared with the general population ($P<.05$).[5]

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Given that gastroparesis is already common in a broad range of patientsit is critical for the health care provider to have a fundamental understanding of this disease and its treatment. This monograph explores the pathophysiology, consequences, diagnosis, and treatment of gastroparesis.

## PATHOPHYSIOLOGY OF GASTROPARESIS

The normal gastric emptying process is controlled through a complex interaction among the autonomic nervous system, the enteric nervous system, and the interstitial cells of Cajal, which act as pacemakers that coordinate the propagation of gastric rhythmic slow waves.[6] Failure at any point in these interactions can result in abnormalities of gastric motility.

When food enters the stomach in normal individuals, the fundus and upper portion of the body relax, accommodating food with little increase in pressure. This relaxation is vagally mediated and uses nitric oxide as a neurochemical effector. Contractions of the antrum and pylorus are controlled by an electrical slow wave generated by the interstitial cells of Cajal. Three to 4 strong peristaltic waves of contraction of about 10 seconds in duration—sometimes referred to as the antral systole—are produced per minute, and these increase in amplitude as they propagate toward the pylorus. The antrum grinds solid food and pumps chyme to the small intestine against pyloric resistance. Solids and liquids empty differently; liquids pass through the pylorus readily, while solids are refluxed backwards, reducing the diameter of solids to 1 to 2 mm—a size that can readily pass through the pylorus. Motility in both the proximal and distal regions of the stomach is controlled by a very complex set of neural and hormonal signals. Nervous control originates from the enteric nervous system as well as parasympathetic (predominantly vagus nerve) and sympathetic nervous systems. A broad range of hormones influences gastric motility, including cholecystokinin (CKK),[7] glucagon-like peptide (GLP)-1,[8] peptide YY (PYY),[9] and amylin.[10]

### *Clinical Causes of Gastroparesis*

Although there are many acute and chronic disorders, as well as medications, that can interfere with the normal neuromuscular coordination of the stomach (Table 1 and 2), gastroparesis is most frequently idiopathic or caused by diabetes or surgery.[11] A small series of 146 patients suggests that the top causes of gastroparesis are 36% idiopathic, 29% diabetic, 14% postgastric surgery, 7.5% Parkinson's disease, 4.8% collagen vascular disorders, 4.1% intestinal pseudo-obstruction, and 6% miscellaneous causes (eg, paraneoplastic syndrome, superior mesenteric artery syndrome, median arcuate ligament syndrome).[1] Gastroparesis has also been seen in patients with quiescent Crohn's disease.[12] Putative idiopathic causes include acute viral-like gastroenteritis (23%), gastroesophageal reflux disease (GERD) and nonulcer dyspepsia (19%), and cholecystectomy. Systemic illnesses may also cause or exacerbate gastroparesis; in particular, delayed gastric emptying may be observed in patients with esophageal, gastric, pancreatic, lung, or breast carcinomas as a result of retroperitoneal nerve invasion.[13]

| Gastroesophageal Diseases | Surgical Procedures |
|---|---|
| Gastroesophageal reflux | Gastrectomy |
| Gastritis (chronic or acute) | Roux-en-Y syndrome |
| Acute gastroenteritis | Vagotomy |
| Atrophic gastritis | Pyloromyotomy |
| Peptic ulcer disease | Pancreatectomy |
| **Neuromuscular Disorders** | Antireflux operations |
| Muscular dystrophy | Combined heart-lung transplantations |
| Parkinson disease | **Trauma** |
| **Systemic Disorders** | Head injuries |
| Diabetes mellitus | Spinal cord injuries |
| Hypothyroidism | **Other Etiologies** |
| Uremia | Idiopathic |
| Chronic liver disease | Medications |
| Anorexia nervosa | Idiopathic pseudo-obstruction |
| **Rheumatologic Disorders** | |
| Scleroderma | |

**Table 1: Causes of Gastroparesis[3]**

| Medications That Delay Gastric Emptying | Medications That Accelerate Gastric Emptying |
|---|---|
| Calcium channel blockers (eg, nifedipine, diltiazem, verapamil, others) | Bulk laxatives |
| Potassium | Diazepam |
| Dopamine | Macrolide antibiotics (eg, erythromycin, clarithromycin, azithromycin) |
| Sucralfate | |
| Aluminum hydroxide | |
| Opiates | |
| Tricyclic antidepressants (eg, imipramine, amitriptyline, desipramine) | |
| L-Dopa | |

**Table 2: Common medications that may affect gastric emptying[3]**

The etiology of gastroparesis is perhaps clearest among patients who have diabetes. In these patients, gastroparesis is associated with the development of autonomic neuropathy, often accompanied by disturbances in autonomic control of heart rate and blood pressure.[14,15] Vagal neuropathy may play a role in this patient population. Data also suggest that interstitial cells of Cajal may be depleted in patients with diabetes, accompanied by decreased expression of nitric oxide synthase and substance P.[16]

Idiopathic gastroparesis may be a result of one or more of these structural abnormalities; however, it is likely that other factors, such as psychological stress, may lower the threshold for symptomatic disease.[1] Notably, approximately one-quarter of patients with idiopathic gastroparesis have a history of acute gastroenteritis and/or viral prodromal symptoms.[17] Among patients who have undergone gastric,

esophageal, or pancreatico-duodenal surgery, gastroparesis probably is a result of anatomical disruption of the vagus nerve.

## DIAGNOSIS OF GASTROPARESIS

Gastroparesis appears to affect relatively younger patients. In a population-based study in Olmsted County, Minnesota, the mean age of patients affected was 45 years, with a mean onset at 33.7 years old.[2] The disease was more common in women (37.8/100,000 persons) than in men (9.6/100,000 persons), however, this gender imbalance might represent differences in health care–seeking behaviors rather than in the fundamental pathophysiology of gastroparesis.[2] As noted previously, gastroparesis is seen in up to 18% of patients with diabetes.[3]

The clinical presentation of gastroparesis is variable. Postprandial nausea (up to 74% of cases), vomiting (53%), abdominal pain (45%), bloating (31%), weight loss (30%), postprandial fullness (23%), and early satiety (23%) can all be part of the symptom complex.[11] In patients with diabetes, abdominal fullness and female gender are predictors of diabetic gastroparesis.[4] Validated scoring instruments are available to help grade the severity of disease.[18]

Adequate nutrition is of paramount importance in gastroparesis. The evaluation of the patient with gastroparesis should begin with an assessment of his or her nutritional status. An unintentional loss of ≥7.5% of usual body weight over a 3-month period suggests significant malnutrition and should trigger intervention (Table 3).[19] It is critical in this assessment to compare the patient's current weight with his or her usual body weight, rather than his or her ideal body weight, which has the potential to over- or underestimate true weight loss.[19] Additionally, it is important to ensure that the patient's actual weight represents a euvolemic weight, that is, neither the result of dehydration or of edema.[20] A dietary history should also be obtained, accounting for changes in appetite, problems with chewing and swallowing, average daily dietary intake, the use of supplemental nutrition, the presence of food intolerances or allergies, and the presence on the medication list of concurrent stool bulking agents, laxatives, and medications known to slow gastric emptying.[3,20] Pertinent laboratory studies will include serum glucose, glycosylated hemoglobin, ferritin, vitamin B12, and 25-OH vitamin D (especially in longstanding gastroparesis or if there is a history of gastrectomy).[20]

| | Significant Malnutrition | Severe Malnutrition |
|---|---|---|
| 1 week | 1% to 2% | >2% |
| 1 month | 5% | >5% |
| 3 months | 7.5% | >7.5% |
| 6 months | 10% | >10% |

**Table 3: Evaluation of weight change over time[31]**

Electrolyte disturbances, weight loss, and nutritional and vitamin deficiencies may occur in patients with gastroparesis, warranting a thorough evaluation for malnutrition and specific deficiencies.[21] Common signs and symptoms of vitamin deficiencies in patients with gastroparesis include bleeding of the gums (vitamin C), visual changes (including night blindness [vitamin A]), and neuropathy or impaired memory and confusion (folate, vitamin B12). Reflux esophagitis may result in dysphagia or odynophagia; altered peristalsis may lead to diarrhea and malabsorption, probably owing to bacterial overgrowth. Dry mucous membranes can also suggest underlying gastroparesis. Difficulties with visual accommodation in bright light, anhydrosis, impotence, dizziness on standing, a scleroderma-like clinical picture, peripheral paresthesias, and focal numbness or weaknesses are also part of the protean manifestations and associations with gastroparesis.[21]

As noted above, the physical exam in suspected gastroparesis should include an assessment for dehydration (eg, orthostasis, pallor, poor skin turgor).[11] The physician should auscultate for obstructive high-pitched or absent bowel sounds; a succussion splash may be heard while shaking the abdomen from side to side more than an hour after a meal. Abdominal palpation may reveal tenderness or an enlarged gastric sac mimicking a mass. It must be noted that diabetic gastroparesis occurs most frequently in patients with diabetes and TRIOpathy (ie, nephropathy, retinopathy, neuropathy). Thus, patients with gastroparesis secondary to diabetes will need screening for findings suggestive of microvascular complications, including retinopathy or autonomic neuropathy, with ophthalmoscopy and peripheral sensory tests.

Concerning diagnostic tests, abdominal imaging is often performed, especially when patients present with abdominal pain or discomfort, to rule out dire etiologies. Endoscopy is mandatory to rule out peptic ulcer disease or malignancy and other causes of mechanical obstruction. Retained food in the stomach after an overnight fast can help in positing an empirical diagnosis of gastroparesis.

Scintigraphy is the reference standard test for the diagnosis of gastroparesis.[21] Following an overnight fast, the amount of food remaining in the stomach is measured after ingestion of a standardized radiolabeled meal (a common standard meal includes eggs, 2 slices of white bread, 30 g strawberry jam, and 0.521 mCi technetium-99m sulfur colloid).[22] Retention is abnormal if ≥90% of the tracer remains in the stomach at 1 hour, ≥60% at 2 hours, or if ≥10% remains in the stomach after 4 hours.[19] While this technique is highly reproducible if the technical standards are followed,[23] many centers do not adhere to a standardized procedure, making interpretation of scintigraphy results challenging.[21] Gastric residual measured at 4 hours after ingestion is the most reliable measurement.

Although scintigraphy with scans up to 4 hours remains the reference standard, other methods for investigating gastric emptying have been explored. Breath testing after ingestion of nonradioactive $^{13}$C-labeled substrates, such as octanoic acid and *Spirulina platensis*, provides a convenient, rapid, radioactivity-free method that may be more broadly accessible, relatively less complex, and significantly less expensive than standard scintigraphy.[24] Breath testing appears to correlate well with

standard scintigraphy, detecting abnormal emptying with a sensitivity of 86% and a specificity of 80%; however, it relies on both normal intestinal absorptive function and normal pulmonary function, and thus may not be valid in some patients.

Capsule telemetry may offer another alternative for the assessment of gastric emptying. The utility of capsule technologies for this indication has been assessed in a number of small trials. In one small study conducted in critically ill patients, a capsule that wirelessly transmitted pH, pressure, and temperature was administered to critically ill patients and healthy volunteers.[25] As expected, gastric emptying time was significantly longer in critically ill patients (median, 13.9 hours) compared with healthy volunteers (mean 3.0 hours; $P<.001$). In another study, a direct comparison between the capsule and a radiolabeled meal in healthy subjects (n=87) and patients with gastroparesis (n=61) found that there was reasonable correlation between the 2 methodologies.[26] The Agency for Healthcare Research and Quality (AHRQ) is currently conducting a comparative effectiveness review evaluating wireless motility capsules versus other diagnostic modalities for the assessment of gastroparesis.[27]

Additional options for the investigation of gastroparesis include transabdominal ultrasound, which has the advantage of being noninvasive and is based on serial measurements of the stomach antrum area before and after a standard meal. However, this methodology is operator-dependent, and it has not been used outside of the research setting.[21] Magnetic resonance imaging has also been employed for the evaluation of gastric physiology in a research setting.[28] Antroduodenal manometry can provide valuable information about the coordination of gastric and duodenal motility. This test can distinguish the anatomical location of the motor dysfunction and help differentiate between processes that are neuropathic versus myopathic in nature, but it is invasive, somewhat cumbersome, and definitely not a first-line diagnostic test.[29]

## *MANAGEMENT OF GASTROPARESIS*

The general objectives of gastroparesis management can be summarized as follows[24,29]:

- Reversal and prevention of dehydration, metabolic disturbances (including hyperglycemia and hypoglycemia), and malnutrition
- Relief of gastrointestinal symptoms
- Treatment of the underlying cause, if possible

The first-line management strategy for gastroparesis encompasses dietary manipulation and administration of antiemetic and prokinetic agents. In more difficult cases, endoscopic injection of botulinum toxin into the pyloric sphincter, gastric electric stimulation, decompressing gastrostomy, and feeding jejunostomy tubes may be considered.

### *Dietary management*

Oral nutrition is preferred in all but the most severe cases of gastroparesis.[20] Only in mild cases, however, will disappearance of symptoms be achieved. Specific dietary guidelines have not been rigorously evaluated for the management of gastroparesis; diet should be individualized based on a dietary history to identify foods that may cause symptoms. However, some general principles can be applied. Meal size should be reduced and meal frequency increased to at least 4 to 6 meals per day, and patients should be instructed to chew food well, maintain adequate fluid intake, and to remain upright after meals.[24,30] A pureed or liquid diet approach may be useful in some patients.[21] Fiber may slow gastric emptying[31]; however, studies have been equivocal regarding the impact of altering the dietary fiber content on gastric emptying.[32-34] Nevertheless, fiber reduction may be an effective starting point for dietary modifications; at the very least, over-the-counter fiber/bulking laxatives should be discontinued. Similarly, fat is a potent inhibitor of gastric emptying[19]; however, many patients are unaffected by fat when supplied in liquid form. In general, altering fat intake should be avoided as first-line dietary management because it provides high-density calories in a small volume.[19]

Although this topic is beyond the scope of this review, some patients with severe gastroparesis may require artificial nutritional support. Modalities and recommendations for artificial nutritional support have been reviewed extensively elsewhere.[21] Briefly, first-line options include slow-pump nasogastric feeding and nasojejunal feeding. In clinical practice, patients may not tolerate the volume required to meet nutritional requirements when supplied directly to the stomach; moreover, nasogastric feeding increases the risk for aspiration.[21] Nasojejunal feeding is the preferred route because it bypasses the malfunctioning stomach, and this recommendation is also supported by the National Institute for Health and Clinical Excellence (NICE).[21] Among patients who benefit from a trial of nasojejunal feeding, percutaneous placement of a jejunal tube may be considered.

### *Prokinetics*

Prokinetics, including the dopamine D2 antagonists metoclopramide and domperidone and the macrolide antibiotic erythromycin, have been the mainstay of pharmacologic treatment for gastroparesis for decades.[24] These agents act by increasing gastric motility and decreasing gastric emptying times. As with other pharmacologic agents used for the treatment of gastroparesis, specific data in this disease state are sparse.

Metoclopramide and domperidone, as dopamine D2 antagonists, also act as antiemetic agents.[24] However, metoclopramide is often poorly tolerated because of acute central nervous system side effects (eg, somnolence, dystonic reactions), and its long-term use should be discouraged because of the risk for extrapyramidal effects (eg, tardive dyskinesia), which have led the FDA to issue a black box warning on this drug.

Domperidone does not cross the blood-brain barrier and, therefore, lacks the neurologic side effects of metoclopramide. Hyperprolactinemia may occur with both agents; thus, patients should be monitored for breast tenderness, galactorrhea, and menstrual irregularities. Erythromycin acts as a potent prokinetic by interacting with motilin receptors, particularly when administered intravenously.[35] However, its

long-term efficacy is limited by tachyphylaxis, probably resulting from downregulation of motilin receptors.[35]

***Antiemetic medications***

Antiemetic medications, such as the phenothiazine derivatives promethazine and prochloroperazine and the $5HT_3$ antagonists ondansetron and granisetron, are useful for symptomatic control of nausea and vomiting, although none of these agents have been specifically tested in gastroparesis.[21] Based on case reports, mirtazapine, a tricyclic antidepressant with activity at the $5HT_3$ receptor, may be effective in some patients with severe gastroparesis.[36,37]

***Novel agents***

Agonists of ghrelin, a peptide hormone naturally produced in the stomach that is an analog of motilin and likely acts as an endogenous appetite-stimulating signal, have shown promise as acute treatments for gastroparesis.[38,39] In a randomized clinical trial, the ghrelin agonist TZP-101 was evaluated in diabetic gastroparesis patents with severe nausea and vomiting.[40] Patients were hospitalized and received 4 indvidual 30-minute infusions of 1 of 6 doses of TZP-101 or placebo daily. Statistically significant improvements over placebo were observed in the 80 mcg/kg group for end-of-treatment changes from baseline in the Gastroparesis Cardinal Symptom Index (GCSI) Nausea/Vomiting subscale (reduction in score of -3.82±0.76) and the GCSI Total score (-3.14±0.78) and were maintained at the 30-day follow-up assessment (-2.02±1.63 and -1.99±1.33). TZP-101 administration was associated with reduction in the proportion of days with vomiting compared with placebo (1.2 days in the active treatment group vs 3.2 days of vomiting in the placebo group out of 4 treatment days).

***Pyloric injection of botulinum toxin***

Botulinum toxin blocks neuromuscular transmission by binding to acceptor sites on motor or sympathetic nerve endings, resulting in local inhibition of acetylcholine release.[41] These agents have been approved for a broad range of indications, and—while not approved for gastroparesis—may represent an option in patients with intractable symptoms.[41] When injected around the pyloric sphincter, sphincter relaxation is presumed to facilitate passage of gastric contents into the duodenum, potentially alleviating symptoms.[21] A recent systematic review and meta-analysis of the utility of intrapyloric botulinum toxin injection for gastroparesis identified [15] reports, of which only 2 were randomized, controlled trials. Significant improvement was suggested only by uncontrolled studies, whereas controlled trials did not support the efficacy of botulinum toxin injection.[42]

***Gastric electric stimulation***

An implantable device (Enterra™) has been developed to provide gastric electrical stimulation by providing high-frequency (12 cycles per minute), low-energy stimulation to the stomach.[43,44] Electrodes are implanted in the serosa of the stomach laparoscopically and connected to a pulse generator implanted in a subcutaneous pocket. It is currently approved by the FDA as a humanitarian use device, and data are limited regarding its efficacy in gastroparesis; however, small studies suggest that it improves quality of life[45] and nutritional status[46,47] and may reduce the risk for hospitalization among patients with gastroparesis.[48]

A prospective study implanted 55 patients with refractory diabetic gastroparesis with the device; after a 6-week period in which the device was turned on, patients were subsequently randomized to consecutive 3-month crossover periods with the device on and off.[47] At 6 weeks, the median reduction in weekly vomiting frequency compared with baseline was 57% ($P<.001$). During the crossover period, however, there was no difference in vomiting frequency between patients who had the device turned on or off during the crossover period. At 1 year, vomiting frequency was significantly lower than baseline (67.8%; $P<.001$), and there were significant improvements in total symptom score, gastric emptying, quality of life, and median days in the hospital.

A recently published long-term (up to 10-year) follow-up study of 221 patients with severe gastroparesis who had received the Enterra™ system found that total symptom scores, hospitalization days, and use of medications were significantly reduced among all patients ($P<.05$); however, 7% of patients had their devices removed because of an infection at the pulse generator site.[49] Because implantation of the device is an invasive procedure, potential benefits of gastric electrical stimulation need to be carefully weighed against the risks.

## CONCLUSIONS

The epidemiologic burden of gastroparesis continues to rise as disease recognition grows and underlying risk factors, such as diabetes, become more prevalent.

Despite the profound impact of gastroparesis on the patient and on health care costs, this condition remains underrecognized, undertreated, and understudied. Gastroparesis remains a key area of need for effective therapeutic tools. An effort to undertake a rigorous approach to studying medical treatments, identifying and using clinically meaningful end points, has recently been initiated with the constitution of expert consortia. While a therapeutic algorithm in gastroparesis remains an elusive goal, there is a need for updated recommendations to help guide approaches to treatment.

The approach to the gastroparesis patient remains guided by the general principles outlined above. However, in the absence of well-designed, randomized, placebo-controlled interventional trials, dietary, pharmacologic, and device-based management of gastroparesis is still based on cautious, often empirical, and individualized decision making.

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# Gastroparesis: Diagnosis and Management

**If you wish to receive acknowledgement of participation for this activity, please complete the post test, evaluation form, and request for credit and fax pages 7-11 to 973-867-3684.**

Please select the one best answer by circling the appropriate letter.

**1. Which of the following prokinetic agents is associated with a risk for tardive dyskinesia?**

***a***. Metoclopramide
***b***. Domperidone
***c***. Erythromycin
***d***. Ketoconazole

**2. Which of the following agents is associated with a risk for hyperprolactinemia?**

***a***. Domperidone
***b***. Erythromycin
***c***. Promethazine
***d***. Ondansetron

**3. In a randomized clinical trial, the ghrelin agonist TZP-101 reduced the proportion of days with vomiting from ____ to _____.**

***a***. 5.2, 1.2
***b***. 3.2, 1.2
***c***. 3.6, 0.8
***d***. 1.2, 0.1

**4. True or false: Randomized, controlled trials of intrapyloric botulinum toxin showed that it significantly improves subjective symptoms and objective gastric emptying in patients with gastroparesis.**

***a***. True
***b***. False

**5. Patients with gastroparesis should be instructed to reduce meal size and increase frequency to at least:**

***a***. 3 to 4 times/day
***b***. 4 to 6 times/day
***c***. 6 to 8 times/day
***d***. 8 to 10 times/day

**6. What percentage of patients with functional dyspepsia present with concomitant symptoms of gastroparesis?**

***a***. 10% to 20%
***b***. 20% to 50%
***c***. 30% to 70%
***d***. 45% to 80%

**7. Which of the following correctly describes initial recommendations for fat and fiber intake in patients with gastroparesis?**

***a***. Both the fiber and fat dietary content should be reduced

***b***. Fiber content should be reduced, but reducing fat content should be avoided

***c***. Fat content should be reduced, but reducing fiber content should be avoided

***d***. Both fat and fiber dietary content should be increased

**8. In patients undergoing scintigraphy, retention should be considered abnormal if ≥____% of the radiolabeled meal remains in the stomach after 4 hours.**

***a***. 90%

***b***. 70%

***c***. 40%

***d***. 10%

**9. What percentage of patients with diabetes has some degree of gastroparesis?**

***a***. 3%

***b***. 12%

***a***. 18%

***b***. 27%

**10. Which of the following is the single most common symptom of gastroparesis?**

***a***. Abdominal pain

***b***. Weight loss

***c***. Postprandial nausea

***d***. Vomiting

# Gastroparesis: Diagnosis and Management

Purdue University College of Pharmacy respects and appreciates your opinions. To assist us in evaluating the effectiveness of this activity and to make recommendations for future educational offerings, please take a few minutes to complete this evaluation form.

## How well did this activity meet the following learning objectives?

| | *This learning objective did (or will) increase/ improve my:* | *High Impact* | *Moderate Impact* | *No Impact* | *Not Applicable* |
|---|---|---|---|---|---|
| • Define gastroparesis and identify its prevalence and incidence | ***Knowledge*** | ❑ | ❑ | ❑ | ❑ |
| | ***Competence*** | ❑ | ❑ | ❑ | ❑ |
| | ***Performance*** | ❑ | ❑ | ❑ | ❑ |
| | ***Patient Outcomes*** | ❑ | ❑ | ❑ | ❑ |
| • Discuss the diagnosis of gastroparesis, including risk factors, symptoms, and diagnostic modalities | ***Knowledge*** | ❑ | ❑ | ❑ | ❑ |
| | ***Competence*** | ❑ | ❑ | ❑ | ❑ |
| | ***Performance*** | ❑ | ❑ | ❑ | ❑ |
| | ***Patient Outcomes*** | ❑ | ❑ | ❑ | ❑ |
| • Describe the contemporary management of gastroparesis, with an emphasis on balancing the risks and benefits of therapy | ***Knowledge*** | ❑ | ❑ | ❑ | ❑ |
| | ***Competence*** | ❑ | ❑ | ❑ | ❑ |
| | ***Performance*** | ❑ | ❑ | ❑ | ❑ |
| | ***Patient Outcomes*** | ❑ | ❑ | ❑ | ❑ |

## Impact of the Activity

- Please indicate which of the following American Board of Medical Specialties/Institute of Medicine core competencies were addressed by this educational activity *(select all that apply)*:

❑ Patient care or patient-centered care
❑ Practice-based learning and improvement
❑ Interpersonal and communication skills
❑ Employ evidence-based practice
❑ Interdisciplinary teams
❑ Professionalism
❑ Quality improvement
❑ Medical knowledge
❑ System-based practice
❑ Utilize informatics
❑ None of the above

- The content of this activity matched my current (or potential) scope of practice.

❑ No _____
❑ Yes, please explain _____

- Was this activity scientifically sound and free of commercial bias* or influence?

❑ Yes _____
❑ No, please explain _____

** Commercial bias is defined as a personal judgment in favor of a specific product or service of a commercial interest.*

# Gastroparesis: Diagnosis and Management

| | *Strongly Agree* | *Agree* | *Disagree* | *Strongly Disagree* | *Not Applicable* |
|---|---|---|---|---|---|
| • The educational activity has enhanced my professional effectiveness in treating patients | ❑ | ❑ | ❑ | ❑ | ❑ |
| • The educational activity will result in a change in my practice behavior | ❑ | ❑ | ❑ | ❑ | ❑ |

• How will you change your practice as a result of participating in this activity *(select all that apply)*?

❑ Create/revise protocols, policies, and/or procedures
❑ Change the management and/or treatment of my patients
❑ This activity validated my current practice
❑ I will not make any changes to my practice
❑ Other, please specify: _____
_____

• What new information did you learn during this activity?

_____
_____
_____

• Please indicate any barriers you perceive in implementing these changes.

❑ Lack of experience
❑ Lack of resources (equipment)
❑ Lack of time to assess/counsel patients
❑ Lack of consensus of professional guidelines
❑ Lack of opportunity (patients)
❑ Lack of administrative support
❑ Reimbursement/insurance issues
❑ Patient compliance issues
❑ No barriers
❑ Cost
❑ Other _____
_____

• If you indicated any barriers, how will you address these barriers in order to implement changes in your knowledge, competency, performance, and/or patients' outcomes?

_____
_____
_____

• Comments to help improve this activity?

_____
_____

• Recommendations for future CME/CPE topics.

_____
_____

**To assist with future planning, please attest to time spent on activity:**

_____
_____

I spent _____ hours on this program.

_____
_____

## REQUEST FOR CREDIT

**If you wish to receive acknowledgement of participation for this activity, please complete this request for credit and fax to 973-867-3684.**

*Please do not use abbreviations.*
*We need current and complete information to assure delivery of participation acknowledgement.*

Degree *(please mark appropriate box and circle appropriate degree):*

❑ MD/DO ❑ PharmD/RPh ❑ NP ❑ PA ❑ RN ❑ Other _____

Full Name *(please print clearly)*
Last Name: First Name: Middle Initial:

Street Address:

City: State or Province: Postal Code:

Phone: – – Ext: Fax: – –

Specialty:

E-mail Address:

**Signature is required to receive statement of credit.**

Signature: _____ Date: _____

**Attestation to time spent on activity is required.**

Purdue University College of Pharmacy designates this enduring material for a maximum of *1.0 AMA PRA Category 1 Credit(s)*™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

❑ I participated in the entire activity and claim *1.0 AMA PRA Category 1 Credit(s)*™.

❑ I participated in only part of the activity and claim _____ credits.