FEDERAL BUREAU OF PRISONS
CLINICAL PRACTICE GUIDELINES
GASTROESOPHAGEAL REFLUX DISEASE (GERD) DYSPEPSIA, AND PEPTIC ULCER DISEASE, NOVEMBER, 2001

PURPOSE

The Federal Bureau of Prisons Clinical Practice Guidelines for Gastroesophageal Reflux Disease (GERD), Dyspepsia, and Peptic Ulcer Disease provide recommendations for the medical evaluation and treatment of federal inmates with heartburn and acid indigestion, upper abdominal pain and discomfort, or suspected peptic ulcer disease.

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DEFINITIONS

Barrett's esophagus is the development of metaplastic columnar epithelium of the esophageal mucosa related to GERD.

Clinician is a physician or midlevel provider.

Dyspepsia is nonspecific pain or discomfort centered in the upper abdomen that may include other gastrointestinal symptoms such as early satiety, bloating, or nausea.

Dysphagia is difficulty in swallowing and in passing food from the mouth via the esophagus to the stomach.

Gastroesophageal Reflux Disease (GERD) is the movement of acid stomach contents into the esophagus that presents clinically as heartburn and acid indigestion.

Heartburn, also termed **pyrosis**, is a retrosternal burning sensation that ordinarily radiates from the epigastrium to the mouth.

Helicobacter pylori (H. pylori) is a Gram-negative spiral bacteria that exclusively infects the gastric mucosa and is a causative factor in peptic ulcer disease.

Hiatus hernia is protrusion of a part of the stomach through the diaphragm via the esophageal opening into the thorax. GERD can occur with or without a hiatal hernia; and patients with hiatal hernias may or may not have GERD.

Nonsteroidal Anti-inflammatory Drug (NSAID)-induced gastropathy is injury to the lining of the stomach from NSAIDs by either their direct topical erosive effects, or by systemic impairment of prostaglandin-dependant mucosal protective mechanisms.

Non-ulcer dyspepsia or functional dyspepsia is dyspepsia that has been thoroughly investigated without identifying a specific cause.

Odynophagia is painful swallowing localized in the anterior chest area, occasionally radiating into the upper chest, arms or back. Odynophagia can mimic anginal symptoms.

Peptic ulcers are focal defects in the protective lining of the gastrointestinal tract associated with the presence of gastric acid but most often caused by *H. pylori* infection and/or the use of NSAIDs.

Regurgitation is the spontaneous return of sour or bitter gastric contents into the pharynx.

Water brash describes hypersalivation associated with an episode of esophageal acid exposure.

Zollinger-Ellison Syndrome is a syndrome of single or multiple gastrointestinal ulcerations that result from benign or malignant islet cell tumors of the pancreas (gastrinomas) that secrete high levels of gastrin.

PROCEDURES

Clinical information covered under "Procedures" is outlined as follows:

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4. CHRONIC DISEASE MANAGEMENT
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5. INMATE EDUCATION

1. INTRODUCTION

Gastroesophageal Reflux Disease (GERD) results from the retrograde movement of stomach contents (particularly gastric acid) into the esophagus creating heartburn symptoms and possible damage to the esophageal lining. GERD can occur from a decrease in lower esophageal sphincter pressure, from delayed gastric emptying, and through the upward displacement of the lower esophageal sphincter such as occurs with a hiatal hernia. Many patients with a hiatal hernia, however, do not have GERD. GERD is increasingly recognized as a chronic condition with potential long term complications that include erosive esophagitis, esophageal ulceration and stricture, Barrett's esophagus, and esophageal adenocarcinoma.

Dyspepsia is a very common nonspecific medical condition of upper abdominal pain or discomfort that is often associated with other gastrointestinal symptoms such as early satiety, nausea, and postprandial abdominal bloating or distention. Dyspepsia can be episodic or persistent and is often exacerbated with eating. A specific cause for dyspeptic symptoms, such as gastroduodenal inflammation and ulcers, GERD, and gastric cancers, can be found in nearly one-half of patients with dyspeptic symptoms. Those patients with dyspepsia without a known cause are considered to have functional or non-ulcer dyspepsia.

Peptic Ulcers are focal defects in the lining of the gastrointestinal tract occurring commonly in the duodenal bulb and the lesser curvature of the stomach. Ulcers may extend through the muscularis mucosae only, through the serosa (perforated ulcer), into a solid organ (penetrating ulcer), or into another hollow organ such as the intestine (fistulizing ulcer). Duodenal ulcers are not associated with cancer, whereas gastric ulcers have a low but significant association with cancer that increases with age. Approximately 90% of duodenal ulcers and nearly 80% of gastric ulcers are caused by chronic infection with *H. pylori.* NSAID usage is responsible for the large majority of peptic ulcers not related to *H. pylori* infection. Serious complications of peptic ulcers include gastrointestinal bleeding, perforation, and pyloric obstruction.

H. pylori is a Gram-negative spiral bacteria that infects the gastric mucosa and causes a chronic gastritis, however, many infected persons with concurrent gastritis never become symptomatic. Gastroduodenal ulcer disease develops in

approximately 15% of infected persons. The specific factors related to *H. pylori* infection that cause progression from chronic gastritis to ulcerative disease are unknown. Eradication of *H. pylori* almost uniformly leads to ulcer healing and markedly reduces the risk of ulcer recurrence. Persons infected with *H. pylori* have a significantly increased risk of low grade gastric lymphomas termed "mucosa-associated lymphoid tissue" (MALT) lymphomas. Treatment of *H. pylori* results in histologic improvement of MALT lymphomas.

NSAIDs cause gastroduodenal ulcerations through both topical and systemic effects. The risk of serious NSAID-related gastrointestinal complications increases with NSAID dosage, age greater than 60 years, prior history of peptic ulcer disease, and the concurrent use of anticoagulants or corticosteroids, however, many persons with NSAID-related ulcers have no apparent risk factors. Despite the dose-related effects of NSAIDs, even very low dose aspirin can cause ulcer disease in certain patients.

2. DIAGNOSIS

Medical History: Inmates initially presenting with upper abdominal pain or discomfort should be interviewed in accordance with the following:

- Assess previous history of GERD, dyspepsia, *H. pylori* infection, PUD, gastric cancer, gall bladder disease, pancreatitis, appendicitis, diverticulosis, irritable bowel disease, prior abdominal surgery, diabetes, coronary artery disease, or other relevant past medical history including previous diagnostic evaluations and treatment interventions for abdominal discomfort.

- Determine if the symptoms of heartburn (retrosternal burning radiating to the neck) and acid indigestion (regurgitation of sour or bitter gastric contents toward the pharynx) are present as well as less common symptoms of GERD including water brash, nocturnal asthma symptoms, and chronic hoarseness or cough. (Pyrosis is exacerbated by lying down, straining, bending over, eating a large meal, and may be relieved by antacids. Classic symptoms of heartburn and acid indigestion are highly sensitive and specific for GERD).

- Characterize abdominal discomfort by determining its rapidity of onset, intensity, location, radiation, frequency, duration, and factors that exacerbate or relieve symptoms such as eating and taking antacids.

- Determine if the symptoms are characteristic of peptic ulcer disease such as a gnawing, boring, epigastric pain that is relieved by taking antacid medications. (The pain associated with duodenal ulcers classically occurs one to three hours after a meal and frequently awakens patients from sleep; the pain associated with gastric ulcers is more variable and may be relieved or exacerbated by eating food. Despite these associations, patient symptoms are neither highly specific nor sensitive for diagnosing peptic ulcer disease).

- Identify other associated symptoms of diagnostic importance such as fever and chills, anorexia, weight loss, nausea, vomiting, diarrhea, constipation, dysphagia, odynophagia, melena, hematochezia, hematemesis, and lightheadedness.

- Assess medication usage, particularly agents contributing to dyspeptic symptoms or gastrointestinal bleeding such as aspirin, NSAIDs, and warfarin; and medications potentially masking a serious abdominal condition such as corticosteroids.

- Quantify alcohol use, tobacco use, and caffeine intake.

Evaluating clinicians should also be aware of the following **diagnostic considerations** when assessing patient histories:

- Dysphagia and odynophagia are not characteristic of uncomplicated GERD or PUD and warrant other diagnostic considerations.

- The abrupt development of severe abdominal pain within minutes suggests an acute event such as a perforated ulcer, perforated viscus, intestinal infarct, ruptured abscess, or dissecting aortic aneurysm.

- Myocardial infarction (MI), particularly an inferior wall MI, can present with symptoms of nausea, vomiting, and vague abdominal pain.

- Pancreatitis is associated with severe constant epigastric pain radiating to the back that is exacerbated by eating and drinking alcohol.

- Biliary colic or cholecystitis is associated with right upper quadrant abdominal pain radiating to the infrascapular area that is precipitated by a heavy meal.

- Irritable bowel syndrome (IBS) presents with cramping abdominal pain and bloating that may be confused with dyspepsia.

Unlike dyspepsia, however, IBS is often associated with abdominal discomfort that is relieved with defecation, and is associated with alternating diarrhea and constipation.

- The pain associated with appendicitis may be diffuse or periumbilical before localizing to the right lower quadrant.

- Patients with pneumonia may present with nonspecific upper abdominal discomfort and pain without pulmonary symptoms.

Physical Examination: The physical examination of inmates with upper abdominal discomfort or pain should include a targeted evaluation of at least the following:

- Measurement of vital signs and weight, including orthostatic blood pressure measurements if gastrointestinal bleeding is suspected

- Examination of sclera for jaundice

- Auscultation of the heart and lungs

- Abdominal examination that includes palpation or localized or rebound tenderness, masses, and organomegaly, and auscultation for bowel sounds and bruits

- Rectal examination and testing for occult blood in the stool whenever gastrointestinal bleeding is suspected

- Examination of skin for rashes and jaundice

The evaluating clinician should be aware of the following **diagnostic considerations** from the physical examination:

- The examination of the abdomen in patients with GERD is usually normal.

- Patients with uncomplicated peptic ulcer disease may have mild tenderness in the epigastrium. Abdominal distention and high pitched bowel sounds may develop with complicated ulcer disease.

- A quiet abdomen with rebound tenderness suggests an acute abdomen that requires emergency evaluation.

- Orthostatic hypotension and tachycardia upon standing in the setting of abdominal pain may indicate significant gastrointestinal blood loss and warrants a timely investigation.

- The presence of fever may indicate an infectious etiology such as appendicitis, cholecystitis, pyelonephritis, pneumonia, or intra-abdominal abscess.

- Tenderness over the gall bladder, particularly with deep inspiration (Murphy's sign) is suggestive of cholecystitis.

Diagnostic studies and procedures: The cause of heartburn and upper abdominal pain and discomfort should be initially assessed from the inmate's history and physical examination. Laboratory screening for anemia, leukocytosis, electrolyte imbalance, hyperamylasemia, hyperbilirubinemia, liver enzyme elevations, and other abnormalities should be pursued on a case by case basis depending on the patient's presentation. Specific diagnostic studies and procedures may also be necessary to establish the diagnosis depending on the inmate's clinical presentation.

- GERD: The diagnosis of GERD can ordinarily be made based on a characteristic symptom presentation and a favorable response to acid suppression therapy. Inmates with GERD symptoms who present with the complications of dysphagia, odynophagia, unintentional weight loss, a history of Barrett's esophagus or esophageal stricture should be referred directly for upper endoscopy. Inmates with classic, uncomplicated GERD symptoms who fail empiric therapy with acid suppression medications should be considered for ambulatory esophageal pH monitoring which is the most sensitive and specific diagnostic test for GERD. Ambulatory pH monitoring is performed by placing a probe transnasally with a pH electrode at the tip to a level in the esophagus 5 cm above the lower esophageal sphincter. Upper endoscopy should also be considered for inmates who remain symptomatic despite acid suppression therapy, however, upper endoscopy is less sensitive than pH monitoring for diagnosing GERD, and is more useful for detecting GERD complications or peptic ulcer disease. Other diagnostic tests such as barium upper gastrointestinal radiography, esophageal manometry, and acid perfusion (Bernstein) tests may be useful for specific patients, but are not routinely indicated when evaluating patients with GERD.

- Dyspepsia: The diagnosis of dyspepsia is a diagnosis of exclusion based on a nonspecific history, negative physical examination findings and normal laboratory studies. A direct referral for upper endoscopy is indicated for inmates with dyspepsia who have a history of gastric surgery or have alarm symptoms such as gastrointestinal bleeding, unintentional weight loss, or protracted nausea, vomiting, or early satiety. Patients older than 50 years of age who fail to respond to a brief course of acid suppression or treatment with a prokinetic agent should

also be referred directly for upper endoscopy since the risk of gastric carcinoma increases significantly with age.

Screening for *H. pylori* infection should be considered selectively for inmates with peptic ulcer symptoms and signs who have failed an initial trial of acid suppression or treatment with a prokinetic agent. Testing for *H. pylori* infection is not recommended if non-ulcer dyspepsia is diagnosed following endoscopy, since treatment is not indicated once non-ulcer dyspepsia is confirmed. Other diagnostic studies, such as biliary ultrasonography and abdominal computed tomography should be considered on a case by case basis depending on the inmate's signs and symptoms.

- Peptic Ulcer Disease: Patients with uncomplicated peptic ulcer disease usually have normal hematologic and chemistry laboratory values. Those patients who develop bleeding ulcers may have a mild leukocytosis, anemia, and azotemia with an elevated blood urea nitrogen:creatinine ratio. Routine screening tests for peptic ulcer disease or associated *H. pylori* infection are not recommended in asymptomatic inmates. Testing for *H. pylori* infection is indicated for inmates with signs and symptoms of peptic ulcer disease, including those taking NSAIDS, selected inmates with dyspepsia who have failed initial therapies, and inmates with gastric MALT lymphoma. Testing for *H. pylori* in asymptomatic inmates taking NSAIDs is not ordinarily indicated.

- ***H. pylori* antibody testing:** *H. pylori* infection can be detected through antibody testing of the serum or whole blood. A positive antibody test indicates either past or current infection with *H. pylori*, since *H. pylori* antibodies usually remain positive after successful treatment. The rate of decline in serum antibodies is highly variable and unpredictable. Antibody tests are frequently the tests of choice for detecting *H. pylori* due to the ease of obtaining specimens and the rapid availability of test results.

- ***H. pylori* stool antigen test:** Detection and eradication of *H. pylori* infection can be measured through the stool antigen test, which is an easier and more rapid method than the urea breath test. Testing for *H. pylori* eradication, however, is not routinely indicated unless the inmate fails to clinically respond to therapy.

- ***H. pylori* urea breath test:** The urea breath test is highly sensitive and specific for detecting *H. pylori* infection. The test is performed by having the patient drink an oral preparation containing carbon-labeled urea.

H. pylori in the stomach metabolizes the urea. The radioactive carbon is absorbed, transported to the lungs, and exhaled. Measurements of radioactive carbon in the exhaled breath indicate the presence of *H. pylori* infection. At the time of testing, patients must not be taking proton pump inhibitors for the past four weeks to avoid false negative test results. The urea breath test is useful for documenting cure of *H. pylori* infection following treatment. Testing for *H. pylori* eradication, however, is not routinely indicated unless the inmate fails to clinically respond to therapy.

- **Upper endoscopy** allows for visualization of the gastrointestinal tract for ulcer disease, erosions and inflammation, active bleeding, and other structural defects. Biopsies are always indicated for gastric ulcers to evaluate for possible malignancy. *H. pylori* can also be detected from gastric biopsy specimens through histologic examination, measurement of urease activity, and through bacterial cultures. Upper endoscopy is indicated as a diagnostic test for peptic ulcer disease in the following circumstances:

- Inmates with upper gastrointestinal bleeding, unintentional weight loss, or protracted nausea, vomiting, or early satiety

- Inmates with previously treated gastric ulcer with recurrent symptoms

- Inmates over 50 years of age with symptoms of dyspepsia who fail a brief trial of acid suppression or treatment with a prokinetic agent

- Inmates who are empirically treated for peptic ulcer disease and *H. pylori* infection but fail to improve clinically

- **Upper gastrointestinal barium radiography** is much less sensitive and specific for diagnosing peptic ulcer disease compared to endoscopy and does not allow tissue sampling. Barium studies should not routinely be used to diagnose peptic ulcer disease, but may be useful for patients who refuse or can not undergo upper endoscopy.

- Serum gastrin levels: Fasting serum gastrin levels are usually normal in patients with peptic ulcer disease, therefore routine screening for hypergastrinemia associated

with Zollinger-Ellison syndrome is not recommended. Patients with recurrent ulcers, ulcers refractory to treatment, multiple ulcers, or ulcers associated with diarrhea should be screened for hypergastrinemia by measurement of several fasting serum gastrin levels. Gastrin levels greater than 1000 pg/mL in association with symptoms of peptic ulcer disease are highly suggestive of Zollinger-Ellison syndrome. Moderate increases in gastrin levels occur frequently with the use of PPIs.

- **Acute abdomen:** Inmates with a suspected acute abdomen should have a complete blood count, urinalysis, stool for occult blood, chest radiograph, and plain and upright abdominal films. Free intraperitoneal air on an upright abdominal film may indicate a perforated ulcer or other perforated viscus. If an inmate has unstable vital signs, marked rebound tenderness or other definitive signs of an acute abdomen, the inmate should be immediately transferred to an appropriate emergency facility for further evaluation.

3. TREATMENT

Inmates with heartburn or upper abdominal pain and discomfort should ordinarily be evaluated and treated in accordance with **Appendix 1, Treatment of Gastroesophageal Reflux Disease (GERD); Appendix 2, Treatment of Dyspepsia, Peptic Ulcer Disease, and *H. pylori;* and Appendix 3, Drug Treatment Options for GERD, and *H. pylori*-associated Peptic Ulcer Disease.**

GERD: The goals of treating GERD are to relieve symptoms and to prevent long term complications. A stepwise treatment approach is recommended, since a certain subset of patients will respond to conservative measures and not require chronic therapy with PPI.

- **Step 1:** Conservative measures are effective in as many as 25% of patients with symptoms of GERD. Step 1 measures include the following:

  - Smoking cessation efforts, caffeine reduction, reduction in fat content of meals, and avoidance of foods that may exacerbate reflux symptoms such as citrus juices, spicy foods, and peppermint

  - Weight reduction as necessary; and avoidance of excessively large meals, particularly when containing fatty foods

- Avoidance of lying down within three hours after completing a meal

- Adjustment in medications that reduce the lower esophageal sphincter pressures as medically feasible, (e.g., theophylline, calcium channel blockers, and nitrates)

- Adjustment in medications that can cause esophageal erosions as medically feasible such as tetracycline, potassium chloride, NSAIDs, and iron supplements

- Trial of antacid therapy

- **Step 2:** Acid suppression or prokinetic therapy should be prescribed if Step 1 measures are ineffective after 3 weeks. Step 2 measures include the following:

- Treatment with an H_2 antagonist for 8 weeks

- Treatment with a prokinetic agent such as metoclopramide can be considered on a case by case basis instead or in addition to an H_2 antagonist, particularly if delayed gastric emptying is considered a contributing factor to GERD. Note: Extrapyramidal symptoms can develop with initiation of metoclopramide; and tardive dyskinesia can develop with long term treatment. Chronic therapy with metoclopramide is not recommended.

- **Step 3:** Proton pump inhibitor treatment (low dose)

- Treatment with a once daily PPI medication taken one hour before a meal provides symptomatic relief in the large majority of patients with GERD. A 4 week trial is ordinarily sufficient to assess for an adequate response. PPI therapy should be initiated as a low daily dose before considering a high dose regimen as outlined in Appendix 3. H_2 antagonists should not be used in combination with PPIs.

- **Step 4:** Proton pump inhibitor treatment (high dose)

- A subset of patients with GERD symptoms will not have complete resolution of symptoms with single daily dosages of PPIs after a 4 week trial. Inmates failing low dose PPI therapy, should be evaluated by ambulatory pH monitoring and/or upper endoscopy to confirm the presence of GERD and investigate other potential diagnoses prior to prescribing a high dose PPI regimen. Subspecialty consultation should be considered if the diagnosis is uncertain.

- **Step 5:** Surgical interventions

  - Subspecialty consultation should be obtained in inmates with GERD refractory to medical therapy before recommending surgery. Specialized surgical procedures can be considered if high dose PPI suppressive therapy is ineffective, poorly tolerated, or if GERD is associated with serious complications despite therapy. Surgical intervention, however, may not prevent the development of adenocarcinoma associated with Barrett's esophagus. Preoperative esophageal manometry is ordinarily required. Simple surgical correction of a hiatal hernia is usually ineffective in relieving GERD symptoms.

Dyspepsia: Treatment of dyspepsia is complicated by the frequent lack of a definitive diagnosis and therapeutic failures. In low risk patients, treatment should begin with lifestyle changes such as smoking cessation, weight loss and avoidance of fatty foods. NSAIDs and other provoking medications should be stopped or adjusted. A trial of antacid medications is ordinarily warranted, but is frequently unsuccessful.

If lifestyle changes and antacids do not control symptoms, an 8 week trial of either acid suppression with a H_2-receptor antagonist, or a prokinetic agent is recommended. The choice of treatment should be based on symptoms and other relevant patient characteristics. Screening and treatment of *H. pylori* infection should be considered selectively for inmates with symptoms compatible with peptic ulcer disease who have failed an initial trial of acid suppression or treatment with a prokinetic agent. A small subset of patients with dyspepsia will have underlying or incipient ulcer disease that will respond to *H. pylori* eradication.

Inmates with dyspepsia refractory to lifestyle changes, acid suppression with H_2 antagonists, treatment with prokinetic agents, or eradication of *H. pylori* infection should be managed on a case by case basis. Patients with mild dyspepsia can frequently be observed and followed for spontaneous resolution or worsening of symptoms. Inmates with dyspepsia associated with GERD should be given a trial of a PPI for 4 weeks. Inmates with persistent symptoms should be referred for upper endoscopy and other diagnostic studies as clinically indicated. Other gastrointestinal disorders, particularly biliary disease, should be considered. The development of weight loss or gastrointestinal bleeding warrants a thorough investigation. In refractory cases where an adequate work-up has failed to identify

a somatic cause of dyspepsia, major depression or an anxiety disorder should be considered and treated as appropriate.

Peptic Ulcer Disease:

- **Nonpharmacologic interventions**: The most effective nonpharmacologic intervention for treating peptic ulcer disease is smoking cessation, since cigarette smoking markedly delays the healing of duodenal ulcers and probably affects the healing of gastric ulcers. Aspirin and NSAIDs should be discontinued whenever feasible. Dietary modifications such as bland diets, avoiding spicy foods, and eating small meals, are ineffective interventions for treating peptic ulcer disease.

- ***H. pylori*-associated ulcers**: Patients with duodenal or gastric ulcers associated with *H. pylori* infection, with or without symptoms, on or off NSAIDs, require treatment to eradicate *H. pylori*. Multiple antibiotic/antisecretory regimens are highly effective in treating *H. pylori* infection associated with peptic ulcer disease as outlined in Appendix 3. Treatment regimens should be selected based on efficacy, potential side effects, adherence concerns, previous treatment history, and the BOP drug formulary. Routine testing of *H. pylori* for antibiotic resistance is not recommended to guide treatment choices. Adherence to the complete treatment regimen is essential to maximize chances for eradication, therefore directly observed therapy on a daily, every other day, or weekly basis should be considered on a case by case basis.

Post-treatment evaluation for persistent *H. pylori* infection in patients with peptic ulcer disease is ordinarily **not** indicated if the patient clinically responds to drug therapy. *H. pylori* testing for eradication by stool antigen testing, upper endoscopy, urea breath testing, or other effective method is indicated for inmates who are persistently symptomatic or have recurrent symptoms, have gastric ulcers associated with early cancer, or have peptic ulcer disease associated with serious complications such as gastrointestinal bleeding or perforation. Testing for *H. pylori* eradication should be performed at least four weeks after completing treatment.

PPI therapy should be continued after treatment of *H. pylori* infection on a case by case basis in patients with peptic ulcer disease. Factors that favor continued PPI treatment include the presence of co-morbid illness, the need for potentially complicating medications such as NSAIDs and anticoagulants, and the severity of previous ulcer-related complications.

- **NSAID-related ulcers**: Patients diagnosed with peptic ulcer while taking NSAIDs should ordinarily be treated in accordance with the following guidelines:

  - NSAIDs should be discontinued whenever possible.

  - *H. pylori* infection, if present, should be treated with a standard regimen.

  - If NSAID treatment is essential, PPI therapy should be continued at least until the ulcer has healed. H_2 receptor antagonists and misoprostol are less effective than PPIs for treating peptic ulcers while patients are taking NSAIDs. Abdominal pain and diarrhea associated with misoprostol also reduces patient adherence to therapy. Once the peptic ulcer has healed, chronic prophylaxis with a PPI should be considered as long as the patient requires anti-inflammatory medications. Substitution of the NSAID with a COX-2 inhibitor is ordinarily not necessary for patients on PPIs who also require chronic NSAID therapy.

- **Zollinger-Ellison Syndrome**: Patients with Zollinger-Ellison syndrome frequently require very high doses of PPIs to control symptoms. Medical management should be provided in consultation with a subspecialist.

- **Idiopathic peptic ulcer disease**: Inmates with peptic ulcer disease of unknown etiology should be treated with PPI therapy and followed clinically. Surreptitious aspirin or NSAID usage should be considered.

- **Invasive interventions**: Surgical therapy is rarely necessary for the management of peptic ulcer disease except in medical emergencies such as uncontrolled hemorrhage, perforation, and pyloric obstruction. Hemorrhage can often be less invasively controlled through endoscopic thermocoagulation or topical injection of a hemostatic agent.

- **Intractable ulcers**: Inmates with confirmed peptic ulcer disease that fails to heal despite an appropriate therapy should be carefully evaluated while considering the following possible contributing factors:

  - If the ulcer is in the stomach, a repeat endoscopy is indicated to evaluate for malignancy

  - Nonadherence to *H. pylori* regimen is common. (If retreatment is considered with a different regimen, the

inmate should be placed on daily directly observed therapy)

- Antibiotic resistance to *H. pylori* (If retreatment is considered, a different regimen should be used; rebiopsy and/or antibiotic sensitivity testing may be helpful, but should be considered on a case by case basis)

- Consider surreptitious NSAID usage

- Consider hypersecretory syndrome such a Zollinger-Ellison syndrome

MALT lymphomas: Treatment of *H. pylori* infection is indicated for patients with gastric MALT lymphomas, since treatment is associated with significant disease regression, and remission when treated in the early stages. Post-treatment confirmation of *H. pylori* eradication is necessary through upper endoscopy, urea breath test, stool antigen testing, or other approved method.

4. CHRONIC MANAGEMENT

GERD: GERD is increasingly recognized as a chronic disease that requires long term management of symptoms and prevention of complications.

- **Evaluations**: Inmates with chronic GERD should be evaluated by a clinician as necessary. Clinicians should assess symptom control and the development of dysphagia, odynophagia, or other symptoms that may suggest disease complications. Certain inmates with GERD will have resolution of symptoms with treatment and will not require maintenance therapy. Other inmates will require chronic PPI suppressive treatment to control their symptoms. Long term PPI usage is both safe and effective and does not require laboratory monitoring. Pharmacists with appropriate training and privileging can monitor inmates with GERD in chronic care clinics.

- **Esophageal stricture**: Patients with longstanding GERD associated with erosive esophagitis are at risk for esophageal stricture, that often presents clinically as a slowly progressive dysphagia, experienced first with food then liquids, without weight loss. Stricture is ordinarily treated with esophageal dilatation and chronic PPI therapy.

- **Barrett's esophagus**: Barrett's esophagus develops from the replacement of the normal squamous cell epithelium of the esophagus above the gastroesophageal junction by metaplastic columnar epithelium. Erosive esophagitis may be confused

visually with Barrett's esophagus, therefore biopsy is essential to establish the diagnosis. Barrett's esophagus is associated with the increasing frequency, severity and duration of GERD symptoms, however, some patients with Barrett's esophagus are asymptomatic. In certain patients with Barrett's esophagus the metaplastic epithelium unpredictably progresses to dysplasia and then to adenocarcinoma. The risk of adenocarcinoma is directly related to the length of the dysplastic epithelium, in centimeters.

The indications for endoscopic surveillance for Barrett's esophagus in patients with GERD are poorly defined. A screening upper endoscopy for inmates over 50 years of age with chronic GERD should be considered. Management of known Barrett's esophagus consists of effectively treating GERD and periodic endoscopic surveillance by a subspecialist to detect and measure dysplastic changes. Endoscopic ablation treatments may be effective for dysplasia. PPI therapy alone will not ordinarily improve histologic changes. Esophagectomy is reserved for patients with histologic changes at high risk for adenocarcinoma.

Dyspepsia: Inmates with chronic dyspepsia should be evaluated on an ongoing basis for symptom pattern changes, particularly the development of alarm symptoms that warrant further evaluation such as gastrointestinal bleeding, unintentional weight loss, and nausea and vomiting. Many patients with dyspepsia have intermittent or chronic dyspepsia that can be effectively managed with symptomatic treatment alone. Pharmacists with appropriate training and privileging can monitor inmates with dyspepsia in chronic care clinics.

Peptic Ulcer Disease:

Inmates with uncomplicated peptic ulcer disease who clinically respond to treatment for *H. pylori* do not require PPI maintenance therapy or ongoing clinician monitoring. Inmates on maintenance PPI therapy for complicated or recurrent peptic ulcer disease associated with *H. pylori* infection should be periodically monitored by a clinician.

Inmates with histories of NSAID-related peptic ulcer disease who require chronic NSAIDs should be monitored closely for recurrent ulcer disease and ordinarily require chronic PPI therapy. Substitution of the NSAID with a cyclooxygenase 2 (COX-2) inhibitor is not usually necessary while the patient is on PPI therapy. If ulcer disease recurs with PPI prophylactic therapy, a subspecialty consultation is warranted.

COX-2 inhibitors should be considered for patients with a history of peptic ulcer disease or gastrointestinal bleeding who no longer require PPI therapy, but who require chronic anti-inflammatory medication.

Patients with Zollinger-Ellison syndrome require ongoing monitoring by a physician for recurrent ulcer disease and ongoing consultation with a subspecialist as necessary.

5. INMATE EDUCATION

Inmates diagnosed with GERD or peptic ulcer disease should receive education from a health care provider on the management and treatment of their condition at the time of diagnosis and periodically during clinician evaluations and during treatments administered by nursing and pharmacy staff. Inmates with chronic disease should be considered for more intensive individual or group educational efforts. Educational resources are attached in Appendices 4-6.

ATTACHMENTS

Appendix 1: Treatment of Gastroesophageal Reflux Disease (GERD)

Appendix 2: Treatment of Dyspepsia, Peptic Ulcer Disease and *H. pylori*

Appendix 3: Drug Treatment Options for GERD and *H. pylori*-associated Peptic Ulcer Disease

Appendix 4: Resources: GERD, Dyspepsia, Peptic Ulcer Disease, and *H. pylori* Infection

Appendix 5: Patient Information: Gastroesophageal Reflux Disease (GERD)

Appendix 6: Patient Information: Peptic Ulcer Disease and *H. pylori* infection

Appendix 7: Provider Self-Assessment: GERD, Dyspepsia, Peptic Ulcer Disease and *H. pylori* Infection.

TREATMENT OF GASTROESOPHAGEAL REFLUX DISEASE (GERD)

DIAGNOSIS
history/physical examination
heartburn/acid indigestion
chest discomfort/ R/O cardiac dx
↓

IDENTIFY HIGH RISK PATIENTS
refer directly for upper endoscopy for dysphagia, history of Barrett's esophagus, GI bleeding
otherwise pursue STEP therapy
↓

STEP 1 - LIFESTYLE CHANGES/ANTACID TX
patient education/ smoking cessation/avoid caffeine
small meals/reduce fat content/weight reduction as indicated
no lying down after eating/no eating 2 hours before bedtime
drug adjustments when appropriate
antacid therapy
↓

STEP 2 - H_2 BLOCKER THERAPY
H_2 antagonist drug trial for 8 weeks
+/- prokinetic agent (long term treatment not recommended)
↓

STEP 3 - PROTON PUMP INHIBITOR DRUG THERAPY (LOW DOSE)
PPI drug trial for 4 weeks (maximize one daily therapy)
↓

STEP 4 - PROTON PUMP INHIBITOR DRUG THERAPY (HIGH DOSE)
first pursue further diagnostic tests to confirm diagnosis
- ambulatory pH monitoring +/- upper endoscopy
high dose PPI therapy if GERD confirmed
↓

STEP 5 - SURGICAL INTERVENTION
Obtain subspecialty consultation for refractory cases
Specialized surgical intervention may be indicated

TREATMENT OF DYSPEPSIA, PEPTIC ULCER DISEASE & *H. PYLORI*

DIAGNOSIS
+ dyspepsia without heartburn or acid indigestion
history/physical examination
laboratory studies as necessary
↓

RISK STATUS ASSESSMENT - IDENTIFY HIGH RISK PATIENTS
any alarm symptoms such as GI bleeding, unexplained anemia, unintentional weight loss
age > 50 who fails to respond to brief trial of H_2 antagonist or prokinetic agent
previous gastric surgery
↓
refer directly for upper endoscopy/stop NSAIDs /biopsy all gastric ulcers
ulcer positive/ *H. pylori* + → tx with *H. pylori* regimen
ulcer negative → tx as non-ulcer dyspepsia (*H. pylori* tx not recommended)
ulcer positive/ *H. pylori* negative → targeted diagnostic workup
↓

PURSUE STEP THERAPY FOR LOW RISK PATIENTS
(at every step - monitor closely for alarm symptoms/refer directly for upper endoscopy as indicated)

STEP 1 - smoking cessation/dietary changes/drug adjustments (stop NSAIDs)/antacid trial
↓

STEP 2 - 8 week trial of H_2 antagonist or prokinetic agent*
(choice dependent on symptom presentation and patient characteristics)
↓

STEP 3 - test for *H. pylori* if ulcer symptoms present and treat if positive
if *H. pylori* negative or no response to treatment
↓

STEP 4 - pursue any or all of the following actions dependent on patient characteristics
- observe and monitor closely for alarm symptoms
- trial of PPI if dyspepsia is complicated by GERD symptoms
- further diagnostic study - upper endoscopy/biliary ultrasound/abdominal CAT scan
- GI subspecialty consultation

* Long term treatment with metoclopramide is not recommended due the abatement of drug efficacy over time and the potential for extrapyramidal side effects and tardive dyskinesia.

Drug Treatment Options for GERD and *H. pylori*-associated Peptic Ulcer Disease

Medications Used in the Treatment of GERD *Please refer to current BOP National Formulary*		
DRUG	**DOSAGE**	**COMMENTS**
ANTACIDS		
Liquid or Tablet Antacid (magnesium, aluminum, or calcium carbonate)	1-2 tablets or 15-30 cc, one hour before meals, two hours after meals, and at HS (additional doses may be supplemented PRN)	Aluminum: constipation Magnesium: diarrhea
H_2 RECEPTOR BLOCKERS (Low dose, over-the-counter)		
Cimetidine	200 - 400 mg BID	Caution: Inhibits cytochrome P-450 system, many drug interactions; gynecomastia
Ranitidine	75 mg BID	Rare side effects
Famotidine	10 mg BID	Rare side effects
Nizatadine	75 mg BID	Rare side effects
PROKINETIC AGENTS		
Metoclopramide	10 mg TID - QID	Avoid long-term use due to potential for extrapyramidal effects (e.g. tardive dyskinesia)
H_2 RECEPTOR BLOCKERS (High dose for GERD)		
Ranitidine	150 mg BID to QID or 300 mg BID	
Famotidine	20 - 40 mg BID	
Nizatadine	150 mg BID	
Continued, next page (Proton Pump Inhibitors)		

Medications Used in the Treatment of GERD *Please refer to current BOP National Formulary*		
PROTON PUMP INHIBITORS		(Tend to be well-tolerated)
Lansoprazole	LOW DOSE: 15 - 30 mg once daily HIGH DOSE: 30 mg BID	
Omeprazole	LOW DOSE: 10 - 20 mg once daily HIGH DOSE: 20 mg BID	Headache is most common side effect.
Pantoprazole	40 mg once daily	Do not split, crush or chew tablets. Higher doses not indicated. (Current indication is for 8 week course for erosive esophagitis)
Esomeprazole	LOW DOSE: 20 mg once daily HIGH DOSE: 40 mg once daily	Contains only one isomer of which Omeprazole contains both d and l. May be emptied into applesauce but not crushed or chewed.
Rabeprazole	20 mg once daily	Do not split, crush or chew tablets.

TREATMENT OF *HELICOBACTER PYLORI* INFECTION

PREFERRED REGIMENS FOR TREATMENT OF H. PYLORI (14 day regimens)			
1	Lansoprazole 30 mg BID	Clarithromycin 500 mg BID	Amoxicillin 1000 mg BID
2	Lansoprazole 30 mg BID	Clarithromycin 500 mg BID	Metronidazole 500 mg BID
3	Ranitidine-Bismuth Citrate (RBC) 400 mg BID	Clarithromycin 500 mg BID	Amoxicillin 1000 mg BID **or** Metronidazole 500 mg BID **or** Tetracycline 500 mg BID
4	Lansoprazole 30 mg **once daily**	Metronidazole 500 mg **TID**	Tetracycline 500 mg QID **and** Bismuth subsalicylate 525 mg QID
5	Ranitidine 150 mg BID or Famotidine 20 mg BID **THEN** Continue the H_2 blocker for an additional two weeks after antibiotics	Metronidazole **250 mg QID**	Tetracycline 500 mg QID **and** Bismuth subsalicylate 525 mg QID

Regimens are all greater than 90% effective in eradicating *H. pylori*; specific regimen should be selected based on adherence issues, patient tolerance, prior treatment regimens, and cost.

RESOURCES
(GERD, Dyspepsia, Peptic Ulcer Disease & *H. pylori*)

National Institute of Diabetes and Digestive and Kidney Disease
National Institutes of Health
TELE: 301-654-3810
WEBSITE: http://www.niddk.nih.gov

Centers for Disease Control and Prevention
National Center for Infectious Diseases
TELE: 1-888-MY ULCER/1-888-698-5237
WEBSITE: www.cdc.gov/nicdod/dbmd/hpylori.htm

American College of Gastroenterology
WEBSITE: http://www.acg.gi.org

American Gastroenterological Association
TELE: 301-654-2055
WEBSITE: http://www.gastro.org/physician.html

Canadian Association of Gastroenterology
WEBSITE: http://www.gi.ucalgary.ca

PATIENT INFORMATION: Gastroesophageal Reflux (GERD)*

What Is Gastroesophageal Reflux?
"Gastroesophageal" refers to the stomach and esophagus. "Reflux" means to flow back or return. Therefore, gastroesophageal reflux is the return of the stomach's contents back up into the esophagus. In normal digestion, the lower esophageal sphincter (LES) allows food to pass into the stomach and closes to prevent food and acidic stomach juices from flowing back into the esophagus. Gastroesophageal reflux occurs when the LES is weak or relaxes inappropriately allowing the stomach's contents to flow up into the esophagus. The severity of GERD depends on LES dysfunction as well as the type and amount of fluid brought up from the stomach and the neutralizing effect of saliva.

What Is the Role of Hiatal Hernia?
Some doctors believe a hiatal hernia may weaken the LES and cause reflux. Hiatal hernia occurs when the upper part of the stomach moves up into the chest through a small opening in the diaphragm (diaphragmatic hiatus). The diaphragm is the muscle separating the stomach from the chest. Recent studies show that the opening in the diaphragm acts as an additional sphincter around the lower end of the esophagus. Studies also show that hiatal hernia results in retention of acid and other contents above this opening. These substances can reflux easily into the esophagus.

Coughing, vomiting, straining, or sudden physical exertion can cause increased pressure in the abdomen resulting in hiatal hernia. Obesity and pregnancy also contribute to this condition. Although considered a condition of middle age, hiatal hernias affect people of all ages. Hiatal hernias usually do not require treatment. However, treatment may be necessary if the hernia is in danger of becoming trapped or is complicated by severe GERD or esophagitis (inflammation of the esophagus). The doctor may perform surgery to reduce the size of the hernia or to prevent strangulation.

What Other Factors Contribute to GERD?
Dietary and lifestyle choices may contribute to GERD. Certain foods and beverages, including chocolate, peppermint, fried or fatty foods, coffee, or alcoholic beverages, may weaken the LES causing reflux and heartburn. Cigarette smoking also relaxes the LES and contributes to GERD symptoms. Obesity and pregnancy can also cause GERD.

What Does Heartburn Feel Like?

Heartburn, also called acid indigestion, is the most common symptom of GERD and usually feels like a burning chest pain beginning behind the breastbone and moving upward to the neck and throat. Many people say it feels like food is coming back into the mouth leaving an acid or bitter taste.

The burning, pressure, or pain of heartburn can last as long as 2 hours and is often worse after eating. Lying down or bending over can also result in heartburn. Many people obtain relief by standing upright or by taking an antacid that clears acid out of the esophagus.

Heartburn pain can be mistaken for the pain associated with heart disease or a heart attack, but there are differences. Exercise may aggravate pain resulting from heart disease, and rest may relieve the pain. Heartburn pain is less likely to be associated with physical activity.

What Is the Treatment for GERD?

Doctors recommend lifestyle and dietary changes for most people with GERD. Treatment aims at decreasing the amount of reflux or reducing damage to the lining of the esophagus from refluxed materials. Avoiding foods and beverages that can weaken the LES is recommended. These foods include chocolate, peppermint, fatty foods, coffee, and alcoholic beverages. Foods and beverages that can irritate a damaged esophageal lining, such as citrus fruits and juices, tomato products, and pepper, should also be avoided.

Decreasing the size of portions at mealtime may also help control symptoms. Eating meals at least 2 to 3 hours before bedtime may lessen reflux by allowing the acid in the stomach to decrease and the stomach to empty partially. In addition, being overweight often worsens symptoms. Many overweight people find relief when they lose weight.

Cigarette smoking weakens the LES; therefore, stopping smoking is very important to reduce GERD symptoms. Antacids taken regularly can neutralize acid in the esophagus and stomach and stop heartburn. Many people find that nonprescription antacids provide temporary or partial relief. Long-term use of antacids, however, can result in side effects, including diarrhea, altered calcium metabolism (a change in the way the body breaks down and uses calcium), and buildup of magnesium in the body. Too much magnesium can be serious for patients with kidney disease. If antacids are needed for more than 3 weeks, a doctor should be consulted.

For chronic reflux and heartburn, the doctor may prescribe medications to reduce acid in the stomach. These medicines include H_2 blockers, which inhibit acid secretion in the stomach, and proton pump (or acid pump) inhibitors, that inhibit an enzyme (a protein in the acid-producing cells of the stomach) necessary for acid secretion.

Other approaches to therapy will increase the strength of the LES and quicken emptying of stomach contents with motility drugs that act on the upper gastrointestinal (GI) tract.

Tips To Control Heartburn

1. Avoid foods and beverages that affect LES pressure or irritate the esophagus lining, including fried and fatty foods, peppermint, chocolate, alcohol, coffee, citrus fruit and juices, and tomato products

2. Lose weight if overweight

3. Stop smoking

4. Elevate the head of the bed 6 inches

5. Avoid lying down until 2 to 3 hours after eating

6. Take an antacid

What If Symptoms Persist?

People with severe, chronic esophageal reflux or with symptoms not relieved by the treatment described above may need a more complete diagnostic evaluation. Doctors use a variety of tests and procedures to examine a patient with chronic heartburn.

For patients in whom diagnosis is difficult, doctors may measure the acid levels inside the esophagus through pH testing. Testing pH monitors the acidity level of the esophagus and symptoms during meals, activity, and sleep.

Endoscopy is an important procedure for individuals with chronic GERD. By placing a small lighted tube with a tiny video camera on the end (endoscope) into the esophagus, the doctor may see inflammation or irritation of the tissue lining the esophagus (esophagitis). If the findings of the endoscopy are abnormal or questionable, biopsy (removing a small sample of tissue) from the lining of the esophagus may be helpful.

Does GERD Require Surgery?
A small number of people with GERD may need surgery because of severe reflux and poor response to medical treatment. Fundoplication is a surgical procedure that increases pressure in the lower esophagus. However, surgery should not be considered until all other measures have been tried.

What Are the Complications of Long-Term GERD?
Sometimes GERD results in serious complications. Esophagitis can occur as a result of too much stomach acid in the esophagus. Esophagitis may cause esophageal bleeding or ulcers. In addition, a narrowing or stricture of the esophagus may occur from chronic scarring. Some people develop a condition known as Barrett's esophagus, which is severe damage to the skin-like lining of the esophagus. Doctors believe this condition may be a precursor to esophageal cancer. Ongoing monitoring of known Barrett's esophagus is necessary so that treatments can be provided when indicated to prevent cancer.

Conclusion
Although GERD can limit daily activities and productivity, it is rarely life-threatening. By understanding the causes of GERD, seeking proper treatment, and following their doctors' instructions, most people will find relief from their discomfort.

*Adapted FROM: NIH Publication No. 94-882, September 1994
e-text posted: February 1998. e-text last updated: April 2000

PATIENT INFORMATION: Peptic Ulcer Disease & *H. pylori**

What Is a Peptic Ulcer?

A peptic ulcer is a sore on the lining of the stomach or duodenum, which is the beginning of the small intestine. Peptic ulcers are common: one in every 10 Americans develops an ulcer at some time in his or her life. The main cause of peptic ulcer is bacterial infection, but some are caused by long-term use of nonsteroidal anti-inflammatory agents (NSAIDs), like aspirin and ibuprofen. In a few cases, cancerous tumors in the stomach or pancreas can cause ulcers. Peptic ulcers are not caused by spicy food or stress.

What Is *H. pylori*?

Helicobacter pylori (*H. pylori*) is a type of bacteria. Researchers recently discovered that *H. pylori* causes almost all peptic ulcers, accounting for up to 80 percent of stomach ulcers and more than 90 percent of duodenal ulcers. *H. pylori* infection is an extremely common infection, however, most infected persons are unaware they are infected and do not develop ulcers. Why *H. pylori* causes ulcers in only a subset of infected persons is unknown. Researchers are not certain how people become infected with *H. pylori*, but they think it may be through food or water.

How Does *H. pylori* Cause a Peptic Ulcer?

H. pylori weakens the protective mucous coating of the stomach and duodenum, which allows acid to get through to the sensitive lining beneath. Both the acid and the bacteria irritate the lining and cause a sore, or ulcer. *H. pylori* is able to survive in stomach acid because it secretes enzymes that neutralize the acid. This mechanism allows *H. pylori* to make its way to the "safe" area--the protective mucous lining. Once there, the bacterium's spiral shape helps it burrow through the mucous lining.

What Are the Symptoms of an Ulcer?

Pain is the most common symptom. The pain usually is a dull, gnawing ache. The pain comes and goes for several days or weeks. It may occur 2 to 3 hours after a meal or in the middle of the night (when the stomach is empty). Pain may be relieved by food. Other symptoms include poor appetite, bloating, burping, nausea, and vomiting. Some people experience only very mild symptoms, or none at all.

The following "ALARM" symptoms are associated with ulcer disease and should always be discussed with a health care provider:

- Sharp, sudden, persistent stomach pain
- Bloody or black stools
- Bloody vomit or vomit that looks like coffee grounds
- Unintentional weight loss
- Nausea, vomiting, or fullness after eating that does not go away with treatment

How Is an H. pylori-related Ulcer Diagnosed?

To see if symptoms are caused by an ulcer the doctor will either consider a trial of medications or perform an endoscopy. An endoscopy is an exam with an endoscope, a thin, lighted tube with a tiny camera on the end. The patient is lightly sedated, and the doctor carefully eases the endoscope through the patient's mouth and down the throat to the stomach and duodenum. This allows the doctor to see the lining of the esophagus, stomach, and duodenum. The doctor can use the endoscope to take photos of ulcers or remove a tiny piece of tissue to view under a microscope.

Diagnosing *H. pylori*

If an ulcer is found on endoscopy, the doctor will test the patient for *H. pylori*. This test is important because treatment for an ulcer caused by *H. pylori* is different from that for an ulcer caused by NSAIDs. *H. pylori* is diagnosed through blood, breath, tissue, and stool tests. Blood tests are the most common. They detect antibodies to *H. pylori* bacteria. Blood is taken at the laboratory through a finger stick.

Breath tests are mainly used after treatment to see if treatment worked, but they can also be used in diagnosis. The test is called a urea breath test. The patient drinks a solution of urea that contains a special carbon atom. If *H. pylori* is present, it breaks down the urea, releasing the carbon. The blood carries the carbon to the lungs, where the patient exhales it. Tissue tests require a biopsy specimen of the ulcer that are stained, examined, or cultured for *H. pylori*. Stool tests measure the presence of a *H. pylori* protein to determine if infection is still present.

How Are H. pylori Peptic Ulcers Treated?

Acid suppressing agents such as H_2-blockers and proton pump inhibitors have been prescribed alone for years as treatments for ulcers. But used alone, these drugs do not eradicate *H. pylori*,

and therefore do not cure *H. pylori*-related ulcers. Effective treatment requires several drugs that together kill bacteria, reduce stomach acid, and protect the stomach lining. Antibiotics are used to kill the bacteria. Agents such as proton pump inhibitors suppress acid production by halting the mechanism that pumps the acid into the stomach. Bismuth subsalicylate, a component of Pepto-Bismol, is used to protect the stomach lining from acid. It also kills *H. pylori*. Treatment usually involves a combination of antibiotics, acid suppressors and stomach protectors. Unfortunately, patients may find multiple drug therapy complicated because it involves taking as many as 20 pills a day. Also, the antibiotics used may cause mild side effects such as nausea, vomiting, diarrhea dark stools, metallic taste in the mouth, dizziness, headache, and yeast infections in women. (Most side effects can be treated by stopping the medication.)

Can *H. pylori* Infection Be Prevented?

No one knows for sure how H. pylori spreads, so prevention is difficult.

POINTS TO REMEMBER

- A peptic ulcer is a sore in the lining of the stomach or duodenum.

- Most peptic ulcers are caused by the *H. pylori* bacterium. None are caused by spicy food or stress.

- *H. pylori* may be transmitted from person to person through contaminated food and water.

- Always wash your hands after using the bathroom and before eating.

- Antibiotics are the most effective treatment for *H. pylori* peptic ulcers.

* Adapted from the National Digestive Diseases Information Clearinghouse, NIH Publication No. 97-4225 October 1997
e-text last updated: 30 January 1998

PROVIDER SELF-ASSESSMENT
(GERD, Dyspepsia, Peptic Ulcer Disease and *H. pylori*)

1. Match the common symptom presentation with the following medical conditions: PANCREATITIS CHOLECYSTITIS, PERFORATED ULCER, IRRITABLE BOWEL SYNDROME, GERD.

A. Sudden severe abdominal pain
B. Substernal burning radiating to the neck
C. Abdominal bloating relieved with defecation
D. Midepigastric pain after eating radiating to the back
E. Right upper quadrant pain after eating radiating to the back

2. Which of the following conditions can initially present with nausea or vomiting or abdominal discomfort?

A. Pneumonia
B. Myocardial infarction
C. Pyelonephritis
D. Pancreatitis
E. All of the above

3. Which of the following is false regarding the diagnosis of patients with abdominal pain?

A. Patients with GERD usually have a normal abdominal examination.
B. Rebound tenderness with a quiet abdomen requires evaluation for an acute abdominal event.
C. Midepigastric discomfort is highly specific for the diagnosis of peptic ulcer disease.
D. Right upper quadrant tenderness to palpation with inspiration may indicate acute cholecystitis.

4. Which of the following statements regarding GERD is false?

A. Heartburn and acid indigestion symptoms are highly sensitive and specific for GERD.
B. Nocturnal asthma symptoms may be indicative of GERD.
C. GERD may require chronic therapy.
D. The absence of a hiatal hernia makes GERD unlikely.

5. Which of the following statements regarding peptic ulcer disease is false?

A. NSAIDs available for IV use can cause peptic ulcers when administered intravenously.
B. The increased risk of peptic ulcers associated with NSAIDs is dose-related.
C. Low dose aspirin does not increase the risk of peptic ulcer disease.
D. Older patients are at an increased risk of NSAID-associated peptic ulcer.

6. Which of the following is false regarding *H. pylori*?

A. Most persons with *H. pylori* infection have chronic gastritis.
B. Eradication of *H. pylori* infection is determined by testing antibody levels following drug treatment.
C. Most peptic ulcers are associated with *H. pylori* infection.
D. Testing for eradication of *H. pylori* infection following treatment is not routinely indicated.

7. Which of the following is an ineffective treatment for peptic ulcer disease?

A. Bland diet/avoiding spicy foods/small meals
B. Smoking cessation
C. Stop NSAIDs
D. *H. pylori* treatment
E. Surgical intervention

8. Most patients with peptic ulcer disease are cured when *H. pylori* is eradicated, but recurrent ulcer disease is common, TRUE or FALSE.

9. Which of the following is false regarding the chronic management of patients with GERD or peptic ulcer disease?

A. Persons with longstanding GERD should be monitored for dysphagia.
B. Most patients with *H. pylori*-related ulcers require long term PPI therapy to prevent recurrence.
C. The incidence of Barrett's esophagus is directly related to the severity and duration of GERD.
D. Gastric ulcers warrant closer monitoring than duodenal ulcers.
E. PPIs are effective in healing and preventing peptic ulcers in patients who must take NSAIDs.

10. Which of the following is not a potential cause for treatment failure of peptic ulcer disease?

A. Nonadherence to *H. pylori* treatment regimen
B. PPI resistance
C. Zollinger-Ellison syndrome
D. Gastric cancer
E. Undisclosed aspirin use

11. An inmate with GERD is persistently symptomatic after being started on 15 mg of lansoprazole in the morning. Which of the following is the most reasonable next step?

A. Maximize single dose PPI therapy
B. Give PPI at night rather than in the morning
C. Add H_2 antagonist to PPI
D. Add antacids to PPI
E. Bernstein test

PROVIDER SELF-ASSESSMENT ANSWERS
(GERD, Dyspepsia, Peptic Ulcer Disease and *H. pylori*)

1. **Matching answers:**

A. Perforated peptic ulcer
B. GERD
C. Irritable bowel syndrome
D. Pancreatitis
E. Cholecystitis

2. **Answer is E**

Vague abdominal symptoms and/or nausea and vomiting may be the presenting complaint in patients with a variety of serious diseases.

3. **Answer is C**

Although patients with peptic ulcers may have a gnawing, boring, pain in the midepigastrium, overall, patient symptom presentations are not highly specific or sensitive for diagnosing peptic ulcer disease.

4. **Answer is D**

Many persons with GERD do not have a hiatal hernia; and many persons with a hiatal hernia do not have GERD.

5. **Answer is C**

The increased risk of peptic ulcer disease associated with NSAID use is dose-related, however, even low dose aspirin increases the risk of ulcer disease. The ulcerogenic effects of NSAIDs are both topical and systemic, therefore parenteral administration of NSAIDs can cause peptic ulcers.

6. **Answer is D**

Most peptic ulcers are associated with *H. pylori* infection and heal with eradication of the organism. Since treatment regimens against *H. pylori* are highly effective, post-treatment testing for *H. pylori* eradication is not ordinarily indicated. Serum antibodies decline unpredictably after treatment of *H. pylori* and may remain positive for several years despite effective therapy.

7. **Answer is A**

All of the treatment interventions with the exception of a bland diet are effective treatments for peptic ulcer disease. Surgery is rarely required since the advent of *H. pylori* therapy and proton pump inhibitors.

8. **Answer is FALSE**

Most patients with peptic ulcers associated with *H. pylori* can be cured of ulcer disease with a low risk of recurrent disease.

9. **Answer is B**

Most inmates who have undergone successful eradication of *H. pylori*, and have a healed ulcer, will not require ongoing medications, including PPIs, nor routine Chronic Care Clinic follow-up.

10. **Answer is B**

PPI resistance does not occur in the setting of *H. pylori* treatment.

11. **Answer is A**

The addition of antacids or H_2 antagonists to PPI therapy offers no additional benefit. PPI treatment should be maximized by giving single dose therapy of 30 mg of lansoprazole before meals, not at bedtime. Twice daily therapy of 15 mg lansoprazole should not be prescribed. Twice daily lansoprazole therapy in dosages greater than 30 mg total daily dose or other PPI regimens with BID dosaging should only be considered if maximal single daily dosaging has failed. The Bernstein test is not routinely recommended for evaluating patients with suspected GERD who fail to adequately respond to PPI therapy. Ambulatory pH monitoring and in some cases upper endoscopy are more effective diagnostic procedures for assessing GERD symptoms on PPIs.