This document has been written for clinicians. The content was developed by the Integrative Medicine Program, Department of Family Medicine, University of Wisconsin-Madison School of Medicine and Public Health in cooperation with Pacific Institute for Research and Evaluation, under contract to the Office of Patient Centered Care and Cultural Transformation, Veterans Health Administration.

Information is organized according to the diagram above, the Components of Proactive Health and Well-Being. While conventional treatments may be covered to some degree, the focus is on other areas of Whole Health that are less likely to be covered elsewhere and may be less familiar to most readers. There is no intention to dismiss what conventional care has to offer. Rather, you are encouraged to learn more about other approaches and how they may be used to complement conventional care. The ultimate decision to use a given approach should be based on many factors, including patient preferences, clinician comfort level, efficacy data, safety, and accessibility. No one approach is right for everyone; personalizing care is of fundamental importance.
Gastroesophageal reflux disease (GERD) is an extremely common condition, affecting nearly one in five U.S. adults at least weekly and nearly one in ten daily.\textsuperscript{1-3} It affects women more commonly than men, and the peak ages are from 30 to 60.\textsuperscript{4}

Symptoms of GERD occur due to esophageal irritation from acidic stomach contents (including pepsin and sometimes bile acids) contacting the esophagus through the lower esophageal sphincter (LES).

A variety of symptoms can occur:

- Retrosternal discomfort (e.g., pain, burning)
- Acid regurgitation
- Nausea and/or vomiting
- Laryngitis
- Cough
- Dental erosions
- Wheezing
- Difficulty swallowing

Normally, the LES only relaxes when one is swallowing food. Otherwise, it has enough tone to limit retrograde flow of acidic contents into the esophagus. With long-term acid exposure, the esophagus may become inflamed (esophagitis) and constrict (stricture), and it can also develop columnar metaplasia (Barrett’s esophagus) or adenocarcinoma.

There are many factors that cause GERD, and these should be systematically evaluated when creating a treatment plan that aims to \textit{cure} rather than just \textit{treat} this disease. The quality of life burden is significant and may be greater than that of congestive heart failure, coronary heart disease, and diabetes.\textsuperscript{5}

Although the LES itself may appear to be the site of dysfunction, it may not play the primary role in GERD pathogenesis. The problem may be downstream, due to increased intra-abdominal pressure (e.g., from obesity, pregnancy, restrictive clothing, ascites). It may also be caused by poor GI motility (gastroparesis) or from forces that challenge normal forward motility (recumbent position, bending over). It may also be due to compromised esophageal mucosal barrier (e.g., from low saliva production). This etiology is often overlooked; one study showed that when acid is infused directly into the esophagus, 88\% of those with GERD were symptomatic, whereas only 15\% of controls without known GERD had symptoms.\textsuperscript{6}
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Studies using proton pump inhibitors (PPIs) and histamine-2 (H2) antagonists show significant benefits in their placebo groups. This supports the notion that the mind has significant potential to affect GERD symptoms. In a large meta-analysis, placebo rates averaged about 20% for pharmaceuticals, indicating that any GERD intervention that one expects benefit will provide a positive response for one in five people independent of the mechanism of action. Creating this positive expectation through a therapeutic clinical relationship can therefore be an important aspect of developing a Personal Health Plan (PHP).

Role of Prevention and Screening

Primary prevention of GERD should be based on health screening and prevention recommendations that apply to all Veterans. Perhaps the most important of these include maintaining a healthy weight, avoiding tobacco products, and limiting excess alcohol consumption. In those with a previous personal history or family history of GERD, it may also be prudent to avoid potentially provocative medications, when possible. These include:

- aminophylline
- anticholinergics
- beta-adrenergics
- calcium channel blockers
- nitrates
- phosphodiesterase inhibitors including sildenafil

Dietary Supplements

**Note:** Please see the module on Dietary Supplements for more information about how to determine whether or not a specific supplement is appropriate for a given individual. Supplements are not regulated with the same degree of oversight as medications, and it is important that clinicians keep this in mind. Products vary greatly in terms of accuracy of labeling, presence of adulterants, and the legitimacy of claims made by the manufacturer.

- **Deglycyrrhizinated licorice** (Glycyrrhiza glabra)—2-4 380 milligrams lozenges before meals. This botanical medicine, like several others on this list, is a demulcent (or mucilaginous). It enhances esophageal mucosal protection. The deglycyrrhizinated form of licorice (DGL), as the name implies, does not contain glycyrrhizin, which has mineralocorticoid actions, such as hypertension, hypokalemia, and edema. Do not use tablets. Rather, use the lozenges, as they can be chewed and swallowed slowly to allow effective contact with the lower esophagus.
- **Slippery elm** (Ulmus fulva)—1-2 tablespoon(s) powder mixed with 1 cup water after meals and before bedtime. This is also a demulcent that is useful for GERD. The root bark powder needs to be carefully titrated with water to ensure a palatable consistency. To enhance flavor, consider adding a small amount of honey or maple.
syrup. This herb has an excellent safety profile, though it has the potential to bind to certain medications and decrease their absorption.8

- **Marshmallow** (*Althea officinalis*)—2-3 teaspoon(s), in divided doses, as an infusion of leaves or root.9 This is another mucilaginous herb with similar properties to slippery elm. Again, this may inhibit the absorption of some medications.8

- **Chamomile** (*Matricaria recutita*)—1 tablespoon dried flowers per cup hot water as a tea 3-4 times daily.10 This botanical has antispasmodic effects on the GI tract, but its use for GERD likely comes from its anti-inflammatory effects.8 Be mindful if one has allergies to plants in the daisy family (*Asteraceae*), as there may be some cross-reactivity.

**Other Systems**

Traditional Chinese medicine (TCM) provides a comprehensive diagnostic and therapeutic framework for treating individuals with diseases such as GERD through lifestyle changes, botanical medicines, and other modalities, such as acupuncture.

Acupuncture may have clinical efficacy for GERD based on three possible mechanisms. Acupuncture may *stimulate GI motility* and *decrease acid secretion* via the vagus nerve and other parasympathetic pathways. Also, acupuncture may *increase esophageal sensory thresholds*, which are known to be decreased in those with GERD. Although only a few studies have investigated the clinical effectiveness of acupuncture on relieving GERD symptoms, several have shown that this modality has predictable effects on GI motility, acid secretion, and pain thresholds in healthy volunteers.6,9,10 One study involving 60 patients showed that weekly acupuncture was equivalent to a pharmacologic regimen of omeprazole and mosapride after 6 weeks of treatment on both subjective and objective measures.13 Acupuncture may also be more effective than doubling the dose of a PPI in those with persistent symptoms on standard-dose PPIs.14

Acupuncture should be considered based on one’s preferences, if it is easily and widely available, and/or if allopathic treatments have proven ineffective. It may also be a valuable tool in helping one to wean off of a proton-pump inhibitor (see below).

**A Note about Chronic PPI Use**

Although pharmaceuticals can be very effective in relieving GERD symptoms, there are increasing concerns of the harmful effects of chronic acid suppression. In our attempts to balance risks and benefits, and to “first do no harm,” these observations should be routinely considered when deciding whether to continue chronic acid suppression therapies:

**Digestion**

- **Incomplete digestion of protein.** Proteins begin to be digested in our stomach directly by hydrochloric acid and indirectly though hydrochloric acid’s activation of pepsin. Pepsin levels fall within a few days of starting a PPI. One hypothesis that may explain increasing rates of eosinophilic esophagitis is that the esophageal
immune system is reacting to more incompletely digested proteins due to decreased activation of pepsin.\textsuperscript{15,16}

- **Decreased absorption of B12,\textsuperscript{17,18} iron (especially nonheme),\textsuperscript{19,20} and calcium.\textsuperscript{21}** Hydrochloric acid often plays a role in removing vitamins from their carrier proteins, making these accessible for assimilation into our body.

**Immunity**

- **Increases in community-acquired *Clostridium difficile* infection.** In one large case-control study, use of PPI and H\textsubscript{2}-antagonists at least doubled rates of this infection (4\% absolute increase with H\textsubscript{2}-antagonist, 15\% absolute increase with PPI).\textsuperscript{22}

- **Increases in community-acquired pneumonia.** In adults, a large cohort study showed one increased case in 226 of those treated with PPIs and 508 of those treated with H\textsubscript{2}-antagonists.\textsuperscript{23} In children, the rate of pneumonia is 10 times higher for those on these medications.\textsuperscript{24}

- **Increases in acute gastroenteritis in children.** Many children are now being treated for reflux-type symptoms. After 2 months of use in young children aged 4-36 months, their risk of developing gastroenteritis more than doubles.\textsuperscript{24}

**Cancer**

- **Enterochromaffin cell hyperplasia.** Chronic acid suppression causes an adaptive increase secretion of gastrin as the body attempts to restore normal gastric acidity. This leads to a hypergastrinemic state that has been shown to be a risk factor for tumor development.\textsuperscript{25}

- **Increased gastric carcinogens.** Lower gastric acidity equates to increased gastric bacterial load. One hypothesis is that this environment may lead to increased conversion of dietary nitrates to nitrites. The nitrites are then converted to the carcinogenic compound N-nitrosamine.

**Fractures**

- **Associations with long-term acid suppression have been found for hip\textsuperscript{26,27} and spine\textsuperscript{28,29} fractures.** This may be related to impaired calcium absorption, as described above.

Many clinicians place people with GERD on medications with no real thought given to underlying causes, which end up never being addressed. Some patients may try to discontinue PPI therapy on their own, only to have a sudden return of symptoms. However, this return of symptoms may be predictable and not necessarily indicative of continuing GERD pathology. When *healthy and asymptomatic* people are given 40 milligrams of pantoprazole for 6 weeks, they will get 10-14 days of GERD symptoms when they stop therapy.
Figure 1. Symptoms of reflux in people without GERD when taking PPIs versus placebo. Blue, dashed = Took PPI; Red, solid = Placebo group. Reprinted by permission from Macmillan Publishers LTD: American Journal of Gastroenterology, copyright 2010.

Tapering Off a PPI

After someone has made appropriate lifestyle changes, and after underlying causes have been addressed, it may be appropriate to a taper off his or her PPI. Rather than simply decreasing the dose over 2-4 weeks, a combination of modalities should be added to maximize one’s chances for success. See the clinical tool, Coming Off a Proton Pump Inhibitor.

Table 1. Summary of Nonpharmaceutical Options for GERD

<table>
<thead>
<tr>
<th>Therapy</th>
<th>Evidence Rating (SORT Criteria)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nearly daily moderate to vigorous exercise for 30-60 minutes, away from mealtime</td>
<td>B</td>
</tr>
<tr>
<td>Food elimination diet</td>
<td>B</td>
</tr>
<tr>
<td>Increase fiber to meet general recommendations</td>
<td>B</td>
</tr>
<tr>
<td>Elevate head of bed (consider only in those with severe symptoms)</td>
<td>B</td>
</tr>
<tr>
<td>Acupuncture</td>
<td>B</td>
</tr>
<tr>
<td>Abdominal breathing</td>
<td>B</td>
</tr>
<tr>
<td>Botanicals (licorice/DGL, slippery elm, marshmallow, chamomile)</td>
<td>C</td>
</tr>
</tbody>
</table>
Relaxation techniques (e.g., meditation, journaling)

<table>
<thead>
<tr>
<th>Level of Evidence Ratings:</th>
</tr>
</thead>
<tbody>
<tr>
<td>A—Based on consistent, good-quality, patient-oriented evidence. (Systematic review or meta-analysis showing benefit, Cochrane review with clear recommendation, high-quality, patient-oriented randomized controlled trial.)</td>
</tr>
<tr>
<td>B—Based on inconsistent or limited-quality, patient-oriented evidence.</td>
</tr>
<tr>
<td>C—Based on consensus, usual practice, opinion, disease-oriented evidence or case series.</td>
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</tbody>
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This clinical tool was written by David Lessens, MD, MPH, integrative medicine family physician at Southcentral Foundation, Anchorage, Alaska. Sections of this clinical tool were adapted from the University of Wisconsin Integrative Medicine Pearls for Clinicians document on Gastroesophageal Reflux Disease prepared by David Kiefer, MD, David Rakel, MD, and Rian Podein, MD.

References

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