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1 Overview

Summary and Quick Facts for Gastroesophageal Reflux Disease (GERD)

- Gastroesophageal reflux disease (GERD) is a chronic condition in which contents of the stomach flow back (reflux) into the esophagus potentially causing symptoms (e.g., heartburn) and injury to esophageal tissue. GERD is one of the most common health conditions of the gastrointestinal tract, and close to 20% of Americans experience heartburn weekly.
- In this protocol, you will learn about the causes of GERD, as well as evidence-based treatment strategies with scientifically studied natural compounds, and specific steps to take to avoid the potential dangers associated with the chronic use of pharmaceutical acid-blocking therapies.
- Education on managing stress, proper diet, physical activity and understanding the causes and progression of GERD has been shown to promote significant improvement in patient perception of illness and well-being.

What is GERD?

Gastroesophageal reflux disease (GERD) is a condition in which stomach contents are refluxed back into the esophagus. The disease can often cause severe discomfort. Prolonged esophageal exposure to the harsh stomach contents and subsequent esophageal damage can also lead to complications including esophageal

cancer.

Many people do not realize that prolonged use of proton-pump inhibitor (PPI) drugs to suppress GERD symptoms can lead to impaired nutrient absorption.

Natural integrative interventions like **raft-forming agents** and **deglycyrrhizinated licorice (DGL) extract** can offer relief for GERD symptoms.

What Increases GERD Risk?

- Obesity
- Food sensitivities (blood tests to detect antibodies against specific foods can be useful for GERD symptoms that fluctuate with diet)
- Smoking

What are the Signs and Symptoms of GERD?

- Heartburn
- Nausea
- Difficulty swallowing and/or “lump” in the throat
- Sleep disturbances
- Respiratory symptoms such as coughing

What are the Conventional Medical Treatments for GERD?

- Antacids to neutralize stomach acid
- Histamine-2 receptor (H2) blockers to prevent secretion of stomach acid
- PPIs to inhibit production of stomach acid
- Gamma-aminobutyric acid B (GABAB) receptor activators to reduce lower esophageal sphincter relaxation
- Surgery to reconstruct the lower esophageal sphincter mechanism

Note: Long-term use of PPIs and other acid-blocking drugs may cause adverse effects. PPIs may impair nutrient absorption and lead to deficiencies in the body. In one study, chronic use of high doses of PPIs and H2 receptor blockers increased the risk of hip fractures significantly.

What Dietary and Lifestyle Changes Can Help Relieve GERD?

- Avoid foods and beverages associated with GERD symptoms including coffee, chocolate, spicy foods, carbonated beverages, and alcohol.
- Quit smoking, as it can increase symptoms.
- Increased body mass can cause pressure on the stomach, increasing stress on the esophageal valve. Obese individuals have shown a 94% increase in GERD symptoms over those with body masses in the normal range. Losing weight may help alleviate symptoms.
- Reduced fat and carbohydrate intake may help alleviate symptoms.
- Avoid eating close to bedtime and sleep with your head elevated.
- Limit use of aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs).

What Natural Interventions May Be Beneficial for GERD?

- **Melatonin**. Melatonin exhibits GI tract protecting abilities and has shown faster relief for GERD symptoms than a commonly-used PPI.

- **Raft-forming agents**. These agents combine gel-forming fibers with an antacid to form a “raft” to block stomach contents from entering the esophagus.
- **Deglycyrrhizinated licorice (DGL) extract**. DGL extract can help heal the delicate tissues of the digestive tract and has shown effectiveness in treating indigestion.
- **Mineral carbonates**. Calcium and magnesium carbonate, and potassium bicarbonate, neutralize stomach acids to decrease irritation of the esophagus.
- Increased intake of **fruits and vegetables**, **fiber**, **zinc**, **vitamins E and C**, **beta carotene**, and dietary **folate** are linked with lower risks of esophageal cancer.

2 Introduction

Gastroesophageal reflux disease (GERD) is a chronic condition in which contents of the stomach flow back (“reflux”) into the esophagus potentially causing symptoms (e.g., heartburn) and injury to esophageal tissue (Patti 2012; Longstreth 2011). GERD is one of the most common health conditions of the gastrointestinal tract, and close to 20% of Americans experience heartburn weekly (Richter 2010, Locke 1997).

Pharmaceutical behemoths rake in nearly **14 billion dollars** annually from the sale of **proton pump inhibitors (PPIs)** in the U.S. alone (Katz 2010) However, long-term use of acid blocking drugs can ***impair nutrient absorption*** and may lead to **deficiencies** with dangerous consequences. For example, chronic, high-dose therapy with *proton pump inhibitors* and *histamine-2 receptor blockers* can significantly increase the risk of **hip fracture** (Corley 2010).

In addition to *robbing* your body of critical nutrients like **calcium**, **magnesium** and **vitamin B12**, PPIs can also cause a ***rebound effect*** when they are discontinued**,** potentially exacerbating GERD symptoms (Khalili 2012; Sheen 2011; Roulet 2012).

Furthermore, conventional treatment strategies call for increasing the dosage or adding *another* acid blocking drug when PPIs fail to relieve GERD symptoms, which occurs in up to **33%** of cases (Fass 2009). Worse yet, as much as **69%** of prescriptions for PPIs are written for *inappropriate indications* (Katz 2010).

In this protocol, you will learn about the causes of GERD, as well as evidence-based treatment strategies with scientifically studied **natural compounds**, and specific steps to take to avoid the potential dangers associated with the chronic use of pharmaceutical acid-blocking therapies.

3 The Esophagus and Digestive Tract

The esophagus conveys ingested material from the mouth to the stomach. It is one of the simpler **regions of the gastrointestinal (GI) tract**; a roughly 8-10 inch muscular tube that runs from the back of the oral cavity (i.e., pharynx), through the chest cavity, and into the abdomen where it joins with the opening of the stomach (i.e., cardia). After ingested materials have traveled down the esophagus, they are emptied into the acidic environment of the stomach for chemical and mechanical digestion.

While the thick cellular layer of the stomach is a suitable barrier against stomach acid, the thinner mucous membrane of the esophagus was not designed to withstand such harsh conditions. To protect the esophagus from the potential back-flow of stomach contents (reflux), a sphincter is located at the junction between the esophagus and stomach, called the gastroesophageal or **lower esophageal sphincter (LES)**. This sphincter, a circular band of thickened muscle, surrounds the lower esophagus and pinches it closed. The LES is usually closed. It opens to allow the passage of swallowed food or drink, a reflex that is triggered by the act of swallowing.

Aiding the closure of the LES is the diaphragm (a wide, flat muscle that helps to expand the lungs during respiration). Internally, the diaphragm separates the chest cavity from the abdomen, and the esophagus passes

through a hole in the diaphragm (called the hiatus) on its way from the mouth to the stomach. The LES is situated near the part of the esophagus that passes through the diaphragm, so that contraction of the diaphragm can reinforce the closure of the sphincter (Kuo 2006).

4 The Development of GERD

Occasionally, increased pressure within the abdomen or a momentary relaxation of the esophageal sphincter can force some stomach contents back into the esophagus. Everyone experiences occasional reflux, which can result from a large meal, physical activity, or reclining after a meal. Other physiological conditions, both normal (pregnancy) and pathological (obesity) can also increase the likelihood of reflux. As long as gastric reflux is occasional, and promptly cleared from the esophagus, there is little risk of damage (Kuo 2006).

Prolonged reflux, however, can present serious health concerns. Repeated exposure of the esophagus to the harsh chemistry of the stomach can have deleterious effects on esophageal tissue (Stefanidis 2010).

It is important to note that while stomach acid is most often associated with reflux disease, there are other compounds, such as bile acids, that may be present in refluxed digestive juices. This is an important consideration in the diagnosis and treatment of GERD, especially when the disease is resistant to acid-suppression therapy. Many patients with treatment-resistant GERD (despite use of acid-blocking pharmaceutical therapy) may have bile in their reflux (Bredenoord 2012; McQuaid 2011). In one study, over half of the patients with reflux symptoms had 24-hour esophageal pH measurements within normal limits (Martinez 2003; Shi 1995). In some cases, stomach acid may partially neutralize damaging alkaline reflux from lower parts of the digestive tract, thus protecting the esophagus. In an animal model of small intestinal reflux, rats on acid-blocking therapy demonstrated more esophageal damage and Barrett's esophagus (see below) than control animals, a finding consistent with some human observational studies (Nasr 2012).

A functional (transient LES relaxation) or mechanical (hypotensive LES) problem of the LES are the most common causes of GERD. Transient relaxation of the LES can be caused by foods (coffee, alcohol, chocolate, fatty meals), medications (beta-agonists, nitrates, calcium channel blockers, anticholinergic drugs), hormones (e.g., progestins), and nicotine (Tutuian 2010).

Allergy *is not a risk for progression to GERD, but it can increase the incidence of a related condition -* ***eosinophilic esophagitis*** **(EE)**. EE is characterized by upper gastrointestinal symptoms (some of which resemble GERD such as food impaction, heartburn, or difficulty swallowing) as well as the presence of *eosinophils* (a type of white blood cell often involved in allergic immune responses) in the esophagus (Arora 2004). Individuals with EE often have normal levels of acidity in the esophagus. Children with EE appear to be unresponsive to acid-blocking therapies. Once thought a rare condition, it is being increasingly recognized as a cause of GERD-like symptoms in adults and, especially children (Gonsalves 2009; Foroutan 2010). Inhaled and food allergens are thought to be responsible for the progression of EE. As such, it is commonly managed by either eliminating the source of the allergen or with steroids (Arora 2004). For individuals with GERD-like symptoms that have been unresponsive to conventional GERD treatments, an **elimination diet** based upon IgE food allergy testing may be useful (Spergel 2005).

Previous evidence indicates that ***food sensitivities*** may contribute to GERD-related irritation of the esophagus (Price 1978). Since immunoreactivity to certain foods is linked with GERD, especially in children (Semeniuk 2009), then using a convenient blood test to assess IgG antibodies against specific foods may prove to be a useful tool for those whose GERD symptoms fluctuate with diet. While clinical studies have yet to evaluate the potential of an elimination diet based upon IgG antibody testing for the relief of GERD symptoms, similar approaches have been successful (Gonsalves 2012).

For more information about food allergy and/or sensitivity testing, refer to the Allergies protocol.

H. pylori Infection Infection with the bacteria *Helicobacter pylori*, which resides in the stomach and is associated with ulcers of the gastric lining, has been observed in up to 40% of GERD cases; though it is unclear whether *H. pylori* infection causes GERD (O'Connor 1999; Ferri 2012). Until recently some experts believed that *H. pylori* infection possibly conferred some protection against development of GERD (Haruma 2007). However, more

recent research suggests that this is likely not the case (Saad 2012; Yaghoobi 2010). Furthermore, upon conducting a review of randomized controlled trials, Saad (2012) and colleagues uncovered a significantly lower incidence of GERD symptoms in patients who had undergone *H. pylori* eradication compared to those who had not.

An Alternative Theory on the Cause of GERD

Hypochlorhydria – Some high-profile alternative medical practitioners suggest that, rather than *too much* stomach acid, it may be *too little* that causes GERD (Wright 2001). The proposed etiology involving *hypochlorhydria* attributes GERD symptoms to refluxed acid, and argues that just because acid is being refluxed does not necessarily mean that there is too much in the stomach to begin with. Proponents of the hypochlorhydria theory believe that inadequate stomach acid reduces lower esophageal sphincter tone, thereby allowing stomach contents to be refluxed giving rise to GERD symptoms.

Hydrochloric acid (HCl) in the stomach activates enzymes that help break down proteins and stimulates other digestive processes. The hypochlorhydria theory of GERD proposes *increasing* stomach acidity to alleviate symptoms, as opposed to lowering it, which is the conventional approach. To do this, alternative practitioners often administer betaine HCl, which delivers additional hydrochloric acid to the stomach. This therapy is sometimes preceded by the Heidelberg test to measure the pH of the stomach.

Although the hypochlorhydria theory lacks support in modern published, peer-reviewed scientific literature, detailed reports of using orally administered hydrochloric acid to improve gastric function are available in early articles (Kern 1926). Also, some innovative modern-day practitioners have noted clinical improvement in GERD patients using this method (Wright 2001).

5 GERD Symptoms and Complications

Aside from **heartburn**, there are several other symptoms associated with GERD that reduce quality of life. These include nausea, hypersalivation (increased saliva production), globus (the sensation of a constant lump in the throat), trouble swallowing, bad breath, and dental erosion (Stanghellini 2004). Sleep disturbances and nocturnal choking are also possible (Kamal 2010). Because of the close proximity of the larynx (the opening of the windpipe) and esophagus, GERD can manifest respiratory symptoms (e.g., including chronic hoarseness, cough, and laryngitis) as well. GERD can be associated with inflammation of lung tissue (pneumonitis), sinusitis, asthma, and middle ear inflection (otitis media) (Amarasiri 2010; Bresci 2010). Recent evidence suggests that GERD may also be associated with idiopathic pulmonary fibrosis (IPF), an incurable lung disease resulting from the deposit of fibrous tissue on the lung surface. The incidence of GERD is high in IPF patients, which places them at risk for aspiration (reflux) of material into the lungs and the subsequent damage as a result (Lee 2010; Fahim 2011).

Prolonged exposure of the esophagus to gastric reflux can result in dramatic alterations to its function. Serious complications of GERD include:

Peptic Stricture. In people with long-term GERD, healing of ulcerations can lead to the deposit of fibrous scar tissue as well as a stricture (i.e., narrowing) of the esophagus (Rosemurgy 2011). Segments of the esophagus with stricture are usually thickened, stiff, and may be shortened. As the esophagus shortens, it can pull the stomach up through the esophageal hiatus, resulting in hiatal hernia (Horvath 2000). The prevalence of peptic stricture among patients with GERD is about 10 to 25% (Hoang 2005). Treatment of severe peptic stricture involves the mechanical dilation of the narrowed region by a stent or balloon combined with acid suppression therapy (Kamal 2010).

Barrett's Esophagus. Barrett's esophagus is a change in the cellular makeup of the mucous membrane of the esophagus. A normal esophagus is lined with a layer of flattened cells (squamous epithelial cells). In Barrett's esophagus, these cells are replaced by a layer of thicker, taller cells (columnar epithelial cells) similar to those

found on the inner surface of the stomach or intestines (Chen 2011). This *reversible* replacement of one differentiated cell type with another mature differentiated cell type is called metaplasia, and is distinct from the cellular transformation that occurs during cancer progression. The main cause of Barrett's esophagus is thought to be an adaptation to chronic acid exposure from reflux esophagitis (Gerson 2002; Toruner 2004). Barrett's esophagus can increase the risk of esophageal cancer. Although endoscopic examination of the esophagus can identify potential tissue changes that are indicative of Barrett's esophagus, a confirmed diagnosis requires a biopsy of the esophageal mucous membrane (Lekakos 2011).

Esophageal Cancer. The two major types of esophageal cancer are esophageal squamous cell carcinoma and esophageal adenocarcinoma. Esophageal adenocarcinoma EAC arises from metaplasia of tissue in the lower part of the esophagus, and is thought to develop as a result of long-term GERD and Barrett's esophagus (Siersema 2007). Two large studies of Barrett's esophagus patients estimate the risk of progression to esophageal adenocarcinoma at approximately 0.27% to 0.4% per person per year (de Jonge 2010; Wani 2011). The risk is highest in men and increases with age, aspirin/NSAID use, smoking, and incidence of hiatal hernia or esophageal dysplasia (de Jonge 2010).

6 Diagnosis

In patients with symptoms that suggest uncomplicated GERD (heartburn and/or regurgitation often occurring after meals and aggravated by lying down or bending over, with relief obtained from antacids), the recommended course of action is treatment for GERD with a trial of acid-suppression therapy. If the patient responds to this initial therapy, then it is reasonable to assume GERD (DeVault 2005).

Self-assessments can be useful in diagnosing uncomplicated GERD. The GERD questionnaire (GerdQ) is a simple, easily interpreted six question assessment of GERD symptom frequency. In one study of 300 patients, GerdQ had 65 percent sensitivity, a result similar to the diagnostic accuracy achieved by gastroenterologists (Jones 2009). Using GerdQ as a patient-tailored diagnostic and therapeutic evaluation tool is beneficial compared with standard approaches to GERD management (Ponce 2011).

Further diagnostic testing is only recommended if the patient does not respond to acid-suppression therapy, presents symptoms suggestive of complicated GERD (e.g., dysphagia), or has been symptomatic long enough to put them at risk for Barrett's esophagus (DeVault 2005).

Tests for GERD may include:

Barium esophagram. Viewing the esophagus via x-ray radiography after swallowing a barium contrast solution can give insight into esophageal motility as well as detect esophageal strictures, ulcers, or severe esophagitis. It is not as sensitive or accurate at diagnosing mild esophagitis or reflux. Compared to newer techniques, it may not be as suitable for the routine diagnosis of GERD (DeVault 2005).

Upper GI Endoscopy. Direct viewing of the esophagus via flexible esophagoscope can identify mucosal breaks, areas of sloughed cells, ulceration, or redness that is distinct from areas of normal mucous membranes. Mucosal breaks are the minimum reliable indicator of GERD (Stefanidis 2010). Esophageal changes indicative of Barrett's esophagus can also be seen with an endoscope. However, a biopsy is required before a definitive diagnosis can be made (Vakil 2006).

Esophageal pH monitoring. **Esophageal pH monitoring** is the current gold standard for diagnosing GERD**.** While a person is upright and mobile, esophageal pH is monitored using a flexible catheter with pH sensor (inserted through the nose and positioned in the lower esophagus), or more recently, a wireless pH capsule attached to the lower esophagus (Roman 2012). Measurements of pH are logged over a 24 hour period (Domingues 2011). Normal esophageal pH is close to 7.0, while a reflux event is recorded as a sudden (< 30 second) drop in pH to below 4.0. One method measures six parameters over the study period including the percentage of time that the esophageal pH is <4 (while upright, reclined, and total), the number of reflux episodes (both total episodes and those > 5 minutes), and the duration of the longest reflux episode. These parameters are then assembled into a

composite score (DeMeester score) where normal is less than 14.7 (Johnson 1974). Unlike endoscopy, esophageal pH monitoring provides direct physiologic measurement of acid in the **esophagus** and is the most objective method to document reflux disease, assess the severity of the disease, and monitor the response of the disease to medical or surgical treatment.

Bilitec. The Bilitec System uses a fiberoptic sensor to detect the presence of bile in reflux. Bile has been implicated in symptomatic reflux that is difficult to manage by conventional acid-suppression therapy (Lazarescu 2008).

Esophageal manometry. Esophageal manometry assesses esophageal and LES function by measuring pressure changes in the esophagus induced by swallowing and peristalsis. A physician passes a pressure-sensing catheter through the nose and esophagus into the stomach. The patient performs a series of 5 mL water swallows, and pressure measurements are made of the peristaltic activity of the esophagus and LES. Since manometry measures esophageal function, it is more suited for diagnosing dysphagsia, or abnormal relaxation of the lower esophageal sphincter (Holloway 2006).

7 Conventional Pharmaceutical Treatment

Acid suppression therapies are the mainstay of pharmaceutical GERD treatment. Acid suppression therapy neutralizes stomach acid or reduces its secretion, minimizing the potential for damage during reflux episodes. Acid suppression therapies include antacids, Histamine-2 receptor blockers, and proton pump inhibitors.

Antacids. Antacids that neutralize stomach acid are the first medication(s) commonly used to provide quick relief for the reflux symptoms of GERD, and are often effective for mild symptoms. Typical antacids include aluminum hydroxide or magnesium hydroxide, calcium carbonate (Tums®), and sodium or potassium bicarbonate.

Histamine-2 receptor blockers (H2 blockers). H2 blockers prevent secretion of stomach acid by inhibiting the action of histamine, which is a stimulus for acid secretion. Examples of H2 blockers include cimetidine (Tagamet®), ranitidine (Zantac®), and famotidine (Pepcid®). H2 receptor blockers are effective for healing only mild esophagitis in 70-80% of patients with GERD and for providing maintenance therapy to prevent relapse. Tachyphylaxis has been observed, suggesting that pharmacologic tolerance can reduce the long-term efficacy of these drugs. Therefore, patients are more likely to develop resistance to the drug, limiting its long-term efficacy (Kim 2004). Still, for mild GERD symptoms, H2 blockers can be an effective treatment.

Proton pump inhibitors. Proton pump inhibitors (PPIs) (e.g., omeprazole, lansoprasole, rabeprazole, pantoprazole, esomeprazole) inhibit stomach acid by preventing the secretion of protons (acid) from acid-producing cells of the stomach. Proton pump inhibitors have been found especially useful when GERD is not well controlled by H2 blockers (Vanderhoff 2002). They are the drug of choice for conventional GERD management (Bruley 2010).

Acid suppression therapies can interfere with the absorption of nutrients that require stomach acid for proper digestion such as non-supplemental iron (Al-Quaiz 2001), vitamin B12 (Ruscin 2002; Dali-Youcef 2009), and dietary calcium (O'Connell 2005).

Acid-suppression-related nutrient deficiencies carry their own health risks. Based upon a study survey, the incidence of iron deficiency anemia secondary to chronic PPI usage has increased up to five-fold (Sarzynski 2011). Elevated homocysteine (a risk factor for cardiovascular disease) has been associated with vitamin B12 deficiency induced by PPIs (Ruscin 2002). Chronic high-dose PPI or H2 blocker usage can more than double risk of potentially deadly hip fracture (Corley 2010; Yang 2006) by impairing calcium absorption and bone metabolism. Impaired calcium absorption can also disrupt calcium balance and contribute to cardiac conduction problems. Individuals on long-term PPI therapy should monitor their iron and B12 status using convenient blood tests to identify possible drug-induced nutrient deficiencies. Homocysteine levels should be assessed as well. Long-term use of acid suppression therapy (drugs) may also be a risk factor for food allergies (Pali-Scholl 2011). In one study, more than 27% of GERD patients tested positive for food allergies; avoiding allergenic foods resulted in significant improvement of GERD symptoms (Pomiecinski 2010).

One of the roles of stomach acid is to provide defense against ingested pathogens. Therefore, reductions in stomach acid due to PPI usage can lower resistance 2-4 fold to intestinal infections from *Salmonella*, *Campylobacter,* and *Clostridium difficile* (Deshpande 2012; Bavishi 2011). Probiotics may prove a useful adjunct to PPI therapy. *Saccharomyces boulardii* (i.e., probiotic yeast) was effective in reducing side effects associated with PPI usage in one study (Song 2010). However, more investigation is needed, as a large 2012 study failed to link PPI therapy to small intestinal bacterial overgrowth (Ratuapli 2012).

Another major problem with PPI drugs is that they are often taken for much longer periods than recommended, which could compound their potential for causing side effects. PPI's are approved for use for 14 day intervals, no more than three times yearly, but many patients take these drugs semi-permanently (Sheen 2011; FDA 2011). Use of PPI's for not more than 14 days at a time and not more than 3 times yearly may reduce the negative side effects of these drugs.

A 2019 article published in *BMJ* provided further evidence of **potential harm associated with PPIs**. The longitudinal observational cohort study followed 157,625 PPI users for 10 years and recorded mortality rates and causes. The researchers found that PPI use was associated with higher mortality rates—specifically due to cardiovascular disease, chronic kidney disease, and certain cancers. The duration of treatment was also related to higher all-cause mortality. The researchers concluded that "heightened vigilance in the use of PPIs may be warranted among those with and—with equal importance—those at risk of these conditions." They recommend PPI use should be limited only to patients where benefits outweigh potential risks, and only for as long as strictly necessary (Xie 2019).

Baclofen. A considerable proportion of GERD patients experience non-acid reflux not alleviated by medications that neutralize stomach acid or inhibit its secretion (van Herwaarden 2002). Treatments that reduce transient lower esophageal sphincter relaxations may be an effective strategy in these individuals.

Baclofen (Lioresal), a medication used to treat muscle spasms by activating gamma-aminobutyric acidB (GABAB) receptors (Mayo Clinic 2017), has now been found to reduce relaxations and increase after-meal pressure in the LES, thereby preventing reflux episodes (Scarpellini 2016; Curcic 2014). A meta-analysis of randomized controlled trials concluded that baclofen effectively reduces the frequency of LES relaxations and the number and duration of reflux episodes (Li 2014). Baclofen is generally used in patients who have an unsatisfactory response to standard treatment. This medication can cause a number of side effects, which has limited its usage in clinical medicine. These side effects include weakness, dizziness, sleepiness, and confusion (Li 2014; Fass 2017).

Surgery. The goal of anti-reflux surgery is the reconstruction of the LES mechanism. This is commonly performed by laparoscopic fundoplication, a minimally invasive technique in which a portion of the stomach (fundus) is wrapped (fully or partially) around the base of the esophagus and sutured into place. The lower portion of the esophagus passes through a small tunnel of stomach muscle (Rosemurgy 2011), thus reinforcing the closure of the LES. Additionally, as the stomach contracts, it constricts the lower esophagus (pinching it shut) instead of forcing acid into the esophagus. There are several surgical techniques for different applications. For example, patients prone to dysphagsia (difficulty swallowing) may benefit more from a laparoscopic fundoplication that only partially wraps the esophagus (Tan 2011). In some cases, these surgeries can be performed as an outpatient procedure (Mariette 2011).

8 Dietary and Lifestyle Approaches to GERD Management

Up to 50 percent of patients with GERD experience persistent symptoms, despite taking PPIs regularly (Dibley 2010). Diet and lifestyle interventions are therefore an important adjunct to standard drug therapy. Education on **managing stress**, proper diet, physical activity, and understanding the causes and progression of GERD has been shown to promote significant improvement in patient perception of their illness and well-being (Dibley 2010).

Some diet and lifestyle modifications commonly suggested for GERD patients include:

Avoid foods and beverages associated with GERD symptoms. Several common dietary components have been associated with increases in GERD symptoms, including

- Coffee (Bhatia 2011)
- Chocolate (Bujanda 2007)
- Spicy foods (Song 2011)
- Carbonated beverages (Hamoui 2006)
- Alcohol (Grande 1996; Song 2011)

Additional foods that may cause symptoms include tomatoes (cooked and raw), milk, cheese, citrus foods, cakes and pastries (Dibley 2010).

Quit Smoking. Smoking increases GERD symptoms by reducing 1) the ability of the LES to remain closed against increases in gastric pressure, and 2) the clearance of reflux from the esophagus (Kaltenbach 2006). The incidence of GERD increases with the duration of smoking. Based upon data from a large population study, long term (> 20 years) daily smoking resulted in a 70% increase in the occurrence of reflux episodes compared to those who have smoked for less than one year (Nilsson 2004).

Lose weight. Increased body mass and abdominal adiposity increases pressure on the stomach and lower esophagus. This can stress the lower esophageal valve, hampering its ability to maintain a seal against gastric reflux. Sustained abdominal pressure can also increase the risk of hiatal hernia (Festi 2009). Based upon a survey of seven studies, overweight individuals averaged a 43% increase and obese individuals a 94% increase in GERD symptoms over individuals with a normal body mass (Hampel 2005). Esophageal adenocarcinoma incidence was more frequent in overweight individuals in most of these studies.

Monitor meal size and macronutrient composition. Dietary fat delays gastric emptying, which may increase the probability of reflux in susceptible patients. High-fat meals are also associated with increased risk of esophageal cancer (De Ceglie 2011). Whereas high-calorie, high-fat meals appear to elicit GERD (Colombo 2002; Fox 2007), reducing fat content in meals has had beneficial effects in some studies (Iwakiri 1996; Penagini 1998). Low carbohydrate (< 20 gram) meals reduced some reflux symptoms in a small trial in obese subjects (Austin 2006). Aside from their direct effects on GERD, limitation of fat, carbohydrate, and total calorie intake are effective methods for weight reduction, which itself is an effective anti-reflux strategy. Weight reduction is also an effective way to positively impact many additional aspects of health and potentially enhance longevity. More information is available in the **Caloric Restriction** protocol.

Avoid eating close to bedtime. GERD patients have long been advised to avoid eating close to bedtime in order to give the stomach adequate time to empty before lying down (DeVault 1999). Clinical studies, however, have had mixed results regarding the minimization of GERD symptoms (Gerson 2009; Orr 1998; Lanzon-Miller 1990).

Elevate the head of the bed while sleeping. Several studies have suggested that raising the head of the bed 8-11 inches, or sleeping on a “wedge”, can reduce the number and duration of reflux episodes (Kaltenbach 2006). This approach uses gravity to help keep stomach contents out of the esophagus. Left lateral recumbency (sleeping on one’s left side) may also reduce GERD symptoms by potentially keeping the LES above the level of the stomach and reducing pressure on the valve (Kaltenbach 2006).

Limit aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs). Some evidence suggests that NSAID use is associated with GERD (Kotzan 2001). NSAIDs exert their anti-inflammatory activity by inhibiting the activity of pro-inflammatory cyclooxygenase (COX) enzymes. However, the COX-1 enzyme is also important for promoting the formation of the protective mucus lining of the stomach.

Results of a prospective cohort study published in early 2021 provided further evidence that lifestyle modifications may help relieve GERD symptoms. An analysis of health questionnaires from over 9,000 middle-aged women who reported experiencing GERD symptoms found that adhering to five “anti-reflux lifestyle factors” may prevent nearly 40% of GERD symptoms, even in those who regularly use PPIs or other anti-reflux medications. The protective lifestyle factors were:

- maintaining a normal body mass index (BMI)
- never smoking
- moderate-to-vigorous exercise at least 30 minutes daily
- no more than two cups of coffee, tea, or soda daily
- a healthy diet

Each lifestyle factor was independently associated with a reduction in GERD symptoms as well, with healthy BMI showing the strongest risk reduction of 31% (Mehta 2021). The authors suggested several mechanisms by which these lifestyle factors may help prevent GERD symptoms, including decreasing lower esophageal sphincter tone and increasing gastroesophageal pressure gradients.

9 Nutrients

Raft-Forming Agents. Raft-forming reflux suppressants have been used to treat GERD for more than 30 years (Hampson 2010). Raft-formers are combinations of a gel-forming fiber (e.g., alginate or pectin) with an antacid buffer (commonly sodium or potassium bicarbonate). When the combination reaches the stomach, chemical reactions cause the release of carbon dioxide bubbles. These bubbles become trapped in the gelled fiber, converting it into a foam that floats on the surface of the stomach contents (hence "raft-forming" agent). Several studies have demonstrated that rafts reduce GERD symptoms by mechanisms independent of acid reduction. They can either move into the esophagus ahead of the stomach contents during reflux (protecting it from exposure) or may act as a barrier to reflux episodes (Mandel 2000). A recent multicenter study of patients with mild to moderate GERD symptoms demonstrated that an alginate-based raft-forming agent was as effective as the PPI omeprazole at reaching an initial heartburn-free period and reducing reflux pain (Pouchain 2012).

The properties of raft-forming agents can be modified by adding calcium salts, which can cross-link fibers and form stiffer gels (Mandel 2000). Raft-formers are most effective when taken after the heaviest meal of the day. If taken with a meal, they can mix with stomach contents and fail to form a "raft" (Mandel 2000).

Melatonin. Melatonin is a hormone most often associated with the sleep cycle, but is found at levels hundreds of times higher in the gut than in the brain (Werbach 2008). Melatonin helps mitigate damage caused by free radicals and inflammatory reactions. Melatonin also helps maintain the mucosal integrity of the oral cavity and esophagus (Brzozowska 2014). Animal studies of melatonin for GERD symptoms have found it to be effective in preventing acid-induced esophageal damage (Konturek 2007). Two human trials have investigated the effect of supplemental melatonin on GERD symptoms. In the first, 176 patients took a multi-nutrient formula that contained 6 mg melatonin. A group of 175 control subjects took a PPI (20 mg omeprazole). The effects were measured by the length of time it took for the patients to become asymptomatic (defined as no heartburn or regurgitation) for 24 hours. All patients in the melatonin group reported improvement in GERD symptoms compared with two-thirds in the PPI group. Relief was reached faster in the melatonin (7 days) versus PPI (9 days) group, with a much lower incidence of side effects (Pereira 2006). The second study compared three groups of nine GERD patients, each on a different regimen (3 mg melatonin, 20 mg omeprazole, or both) to a group of healthy control subjects. Heartburn and gastric pain decreased after four weeks and completely resolved after eight weeks in all treatment groups. However, only the two melatonin groups had significant improvements in lower esophageal sphincter function (Kandil 2010).

Deglycyrrhizinated Licorice (DGL) Extract. Licorice root is a time-honored treatment for digestive ulcers, and modern research continues to confirm its ability to heal the tissues of the digestive tract. Some of the mechanisms behind this remarkable ability are now well understood. Compounds from the licorice plant increase the concentration of prostaglandins at the site of erosive lesions, causing increased mucous secretion and cell proliferation to aid healing. Licorice can also inhibit production of pro-inflammatory cytokines such as

interleukins, tumor necrosis factor, and nuclear factor kappa-B; and is a powerful oxidative stress modulator. These properties contribute to its ability to protect the delicate lining of the gastrointestinal tract (Baker 1994; Furusawa 2009; Asl 2008; Aly 2005).

A compound found in unrefined licorice root, glycyrrhizin, may cause side effects in high doses including bloating, high blood pressure, low blood potassium levels, hormonal changes, and diarrhea. When these compounds are removed from licorice root, the product is called *deglycyrrhizinated licorice*, or DGL. DGL retains the gastrointestinal healing properties of the licorice while avoiding most side effects (Larkworthy 1975; Isbrucker 2006).

Research has shown that DGL extract is an effective treatment for indigestion. In a randomized controlled trial, 50 subjects with functional dyspepsia were randomized to receive a placebo or 75 mg of a patented DGL extract twice daily for 30 days. At 15 and 30 days, subjects taking the GutGard reported significant decreases in total symptom scores compared with those taking placebo. The GutGard recipients also showed significant improvement on a standardized dyspepsia assessment index compared with placebo recipients. GutGard was found to be safe and was well tolerated by all subjects (Raveendra 2012).

Mineral carbonates (calcium, magnesium, and potassium). Calcium and magnesium carbonate, and potassium bicarbonate, neutralize stomach acidity and have been used in antacid preparations for many years (Maton 1999; Gold Standard 2002). Magnesium and calcium carbonate interact with hydrochloric acid in the stomach to form chloride salts, water, carbon dioxide, hydrogen, and other benign products (GCSE Bitesize 2014; The American Society of Health-System Pharmacists 2016).

By neutralizing stomach acid, **antacid mineral carbonates** decrease irritation of the delicate lining of the gastrointestinal tract. Antacids also inhibit the activity of the gastric enzyme pepsin, and this action may also protect against damage to ulcerated or eroded gastrointestinal lining (Gold Standard 2009). Importantly for those suffering from GERD, antacids such as calcium and magnesium carbonate neutralize acid *in the esophagus*; and chewable calcium carbonate has a relatively long duration of action. Chewable calcium carbonate has demonstrated an ability to improve contraction of the esophagus, resulting in increased clearance of acid (McRorie 2014; Robinson 2002; Rodriguez-Stanley 2004). Calcium carbonate has a rapid onset of action and is capable of relieving GERD symptoms in minutes (Robinson 2002).

Protecting against Barrett's Esophagus and Esophageal Cancer

GERD increases the risk of metaplastic (i.e., transformation of tissue) events that lead to Barrett's esophagus, which in turn significantly increases the risk of esophageal cancer. Therefore, the most effective way to reduce the risk of these two serious conditions is to control the symptoms of GERD. However, the following additional considerations may be beneficial as well.

Several observational studies have examined the effects of dietary patterns on the incidence of Barrett's esophagus or esophageal cancer (independently of GERD). Some foods and supplements appear to reduce cancer and metaplasia risk.

Total fruit and vegetable intake has been associated with reductions in the risk of esophageal adenocarcinoma in some studies (Chen 2002; Navarro Silvera 2008; Navarro Silvera 2011). It has been noted that risk reductions associated with citrus fruits as well as yellow, brassica, or raw vegetables were consistently positive (Gonzalez 2006; Steevens 2011; Chen 2002). Strawberries, due to their powerful antioxidants, have also piqued the interest of researchers looking for compounds able to protect esophageal tissue. In order to test the hypothesis that strawberries might protect against esophageal cancer, scientists administered freeze-dried strawberry powder to 75 patients with precancerous esophageal lesions for six months. At a dose of 60 grams daily, freeze dried strawberry powder improved the appearance of the esophageal tissue under microscopic examination. Moreover, several inflammatory markers were reduced, including a 63% reduction in cyclooxygenase-2 (COX-2) activity and a 62% reduction in Nf-kB activity. The investigators remarked that "*Our present results indicate the potential of freeze-dried strawberry powder for preventing human esophageal cancer*" (Chen 2012).

Fiber from cereal or whole grain was generally associated with reduced risk of esophageal cancer (Chen 2002;

Mayne 2001; Terry 2001; Navarro Silvera 2008). On the other hand, increased consumption of animal protein, saturated fat, and dietary cholesterol consistently led to increased risk of esophageal cancer (Mayne 2001; Navarro Silvera 2008).

Vitamins C, E, beta carotene (Mayne 2001; Bollschweiler 2002; Kubo 2007; Carman 2009), and dietary folate (Mayne 2001; Ibiebele 2011; Bollschweiler 2002) appear to confer a reduction in esophageal cancer risk in the majority of studies. Likewise, general supplement (i.e. multivitamin) usage was associated with risk reduction in one population study (Dong 2008).

Several fruits and vegetables contain a powerful polyphenol (antioxidant) called ellagic acid. It exerts cellular protection in a variety of settings and is well-documented in animal studies as an inhibitor of esophageal cancer as well as aiding in ulcer healing (Whitley 2005; Beserra 2011).

Zinc, a trace mineral essential to many biological processes, may protect against esophageal cancer (Grosvenor 1995). One observational study evaluated serum zinc levels in 60 people with esophageal or gastric cancer and 120 healthy controls. People with esophageal cancer had significantly lower serum zinc levels. This study suggests lower levels of zinc in the body correlate with development of esophageal cancer (Hashemi 2017). In a recent laboratory study on human cell lines, the addition of zinc to the cell culture significantly inhibited proliferation of squamous cell esophageal cancer cells. Nonmalignant esophageal cell lines did not display this significant inhibitory response to zinc. Also, zinc's ability to inhibit the proliferation of esophageal cancer cells was reversible with a zinc chelator. The investigators identified modulation of calcium signaling as a possible mechanism by which zinc mediated this anti-cancer effect (Choi 2017). Importantly, proton pump inhibitors, which are often overused by people with GERD, can impair zinc absorption and reduce stores of zinc in the body (Farrell 2011), highlighting the importance of adequate zinc intake in this group.

Various other dietary constituents have been investigated in cell culture or animal models of esophageal cancer with positive results. These include sulforaphane (from broccoli) (Qazi 2010), vitamin E with N-acetyl cysteine (Hao 2009), proanthocyanidins (from apples) (Pierini 2008) and cranberries (Kresty 2008). Betaine (trimethylglycine) intake was associated with a reduction in Barrett's esophagus in one study (Ibiebele 2011).

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