Are Lifestyle Measures Effective in Patients With Gastroesophageal Reflux Disease?

An Evidence-Based Approach

Tonya Kaltenbach, MD; Seth Crockett, MD; Lauren B. Gerson, MD, MSc

Lifestyle modifications are first-line therapy for patients with gastroesophageal reflux disease (GERD). We applied an evidence-based approach to determine the efficacy of lifestyle measures for GERD management. We used PubMed and Ovid to perform a search of the literature published between 1975 and 2004 using the key words heartburn, GERD, smoking, alcohol, obesity, weight loss, caffeine or coffee, citrus, chocolate, spicy food, head of bed elevation, and late-evening meal. Each study was reviewed by 2 reviewers who assigned one of the following ratings: evidence A, randomized clinical trials; evidence B, cohort or case-control studies; evidence C, case reports or flawed clinical trials; evidence D, investigator experience; or evidence E, insufficient information. We screened 2039 studies and identified 100 that were relevant. Only 16 clinical trials examined the impact on GERD (by change in symptoms, esophageal pH variables, or lower esophageal sphincter pressure) of the lifestyle measure. Although there was physiologic evidence that exposure to tobacco, alcohol, chocolate, and high-fat meals decreases lower esophageal sphincter pressure, there was no published evidence of the efficacy of dietary measures. Neither tobacco nor alcohol cessation was associated with improvement in esophageal pH profiles or symptoms (evidence B). Head of bed elevation and left lateral decubitus position improved the overall time that the esophageal pH was less than 4.0 (evidence B). Weight loss improved pH profiles and symptoms (evidence B). Weight loss and head of bed elevation are effective lifestyle interventions for GERD. There is no evidence supporting an improvement in GERD measures after cessation of tobacco, alcohol, or other dietary interventions.

Gastroesophageal reflux disease (GERD) is characterized by symptoms of substernal burning or acid regurgitation produced by the abnormal reflux of gastric contents into the esophagus.1 According to US population surveys, 44% of all Americans experience heartburn at least once per month, 14% at least once per week, and up to 7% daily.2,3 The major mechanism for GERD is transient relaxation of the lower esophageal sphincter (LES).5 Recommendations for lifestyle modifications are based on the presumption that certain foods, body position, tobacco, alcohol, and obesity contribute to a dysfunction in the body’s antireflux defense system. Accordingly, the American College of Gastroenterology recommends the use of lifestyle changes, including elevation of the head of the bed (HOB); decreased fat, chocolate, alcohol, peppermint, and coffee intake; cessation of smoking; and avoiding recumbency for 3 hours postprandially, in addition to antireflux medical treatment.1 However, the evidence to support such lifestyle modification recommendations has not been well substantiated. The purpose of this study is to apply an evidence-based approach to

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the literature to determine which lifestyle measures are most likely to benefit patients with GERD.

METHODS

We performed a systematic review of the English-language literature relating to human subjects published between 1975 and 2004 using PubMed and Ovid to obtain data pertaining to the effects of lifestyle modifications on GERD. We included the following key words in the search: lifestyle modification, heartburn, GERD, reflux, smoking, alcohol, obesity, weight loss, caffeine or coffee, citrus, chocolate, mint, carbonated beverages, spicy food, fatty foods, head of bed elevation, and late-evening meal.

To be included in the study, trials had to contain a lifestyle intervention and outcomes of GERD measures, including heartburn symptoms, ambulatory esophageal pH monitoring, and esophageal manometric variables. Studies of the physiologic effects of lifestyle interventions only are included as introductory evidence for each lifestyle intervention.

We applied an evidence-based approach to the literature using a standard scoring system (Table 1). After 2 of us (either T.K. and L.B.G. or S.C. and L.B.G.) independently reviewed each study, evidence-based ratings of A through E were assigned. A consensus was obtained through a second review if there was a difference of opinion on initial review.

RESULTS

Of 2039 trials screened for relevancy, 100 were included in this analysis. Only 16 trials examined the impact of lifestyle changes on GERD variables. The subsections that follow describe the physiologic evidence of lifestyle interventions on GERD measures (Table 2), followed by the available evidence on cessation of the lifestyle measure and the impact on GERD variables (Table 3).

SMOKING

Cigarette smoking has been associated with symptomatic GERD. Questionnaires have reported higher rates of reflux symptoms in tobacco smokers compared with nonsmokers.6,9 Multivariate analysis from a case-control study10 showed that duration of smoking was associated with increasing reflux symptoms, with an odds ratio (OR) for reflux of 1.7 (95% confidence interval [CI], 1.4-2.0; \( P<.001 \)) in daily smokers with a greater than 20-year tobacco use history compared with those who smoked daily for less than 1 year. Cigarette smoking has been shown to prolong acid clearance and decrease baseline LES pressure (LES).96,97 with an abrupt decline in LES at the start of smoking that returns to normal within minutes of completion of the cigarette.96 Abrupt increases in intra-abdominal pressure, such as with coughing or deep inspiration, have been associated with reflux symptoms in smokers.11 Studies12-14 that examined acid perfusion (using the Bernstein test) or esophageal \( \text{pH} \) have reported conflicting associations; for example, despite having more “reflux episodes,” smokers (compared with nonsmokers) do not show increased esophageal acid exposure time.15

No improvement was shown in 3 case-control studies (evidence B) that evaluated the effect of tobacco cessation on GERD outcomes. One study15 compared a cohort of 30 healthy volunteers (15 smokers and 15 nonsmokers) with 10 smokers with GERD and found that smokers had more reflux episodes than nonsmokers but that 24-hour smoking cessation did not reduce the total time that the esophageal \( \text{pH} \) was less than 4.0. Another study87 showed that 1-day cessation of smoking decreased the number of daily reflux episodes but did not significantly affect total esophageal acid exposure. On the contrary, Kadakia et al.88 after their evaluation of smokers with GERD who abstained from smoking for 48 hours, concluded that smoking significantly increased distal esophageal acid exposure. Although tobacco use has been associated with an adverse effect on the LES, the evidence to date does not support an improvement in GERD after cessation of tobacco use.

ALCOHOL

Alcohol consumption may precipitate GERD by increasing acid secretion through gastrin stimulation, reducing LES, increasing spontaneous LES relaxations, and impairing

Table 1. Evidence Levels

<table>
<thead>
<tr>
<th>Evidence A</th>
<th>Evidence B</th>
<th>Evidence C</th>
<th>Evidence D</th>
<th>Evidence E</th>
</tr>
</thead>
<tbody>
<tr>
<td>One or more well-designed randomized controlled trials, nonrandomized controlled trials, or uncontrolled clinical trials</td>
<td>Cohort or case-control studies</td>
<td>Case reports, flawed clinical trials, population studies</td>
<td>Expert or investigator opinion</td>
<td>Insufficient evidence or trials with significantly conflicting data</td>
</tr>
</tbody>
</table>

Table 2. Summary of Physiologic Evidence

<table>
<thead>
<tr>
<th>Factor</th>
<th>Trials, No.</th>
<th>Lowered LES P</th>
<th>Worsened pH</th>
<th>Worsened Symptoms</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco</td>
<td>12</td>
<td>B</td>
<td>B</td>
<td>B</td>
<td>6-17</td>
</tr>
<tr>
<td>Alcohol</td>
<td>16</td>
<td>No effect (B)</td>
<td>B</td>
<td>B</td>
<td>3,6,9,10,16-27</td>
</tr>
<tr>
<td>Obesity</td>
<td>24</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>28-51</td>
</tr>
<tr>
<td>Coffee and caffeine</td>
<td>14</td>
<td>E</td>
<td>E</td>
<td>No effect (C)</td>
<td>10,16,17,52-62</td>
</tr>
<tr>
<td>Chocolate</td>
<td>2</td>
<td>B</td>
<td>B</td>
<td>E</td>
<td>63,64</td>
</tr>
<tr>
<td>Spicy foods</td>
<td>2</td>
<td>E</td>
<td>E</td>
<td>C</td>
<td>65,66</td>
</tr>
<tr>
<td>Citrus</td>
<td>3</td>
<td>No effect (B)</td>
<td>E</td>
<td>C</td>
<td>52,66,67</td>
</tr>
<tr>
<td>Carbonated beverages</td>
<td>2</td>
<td>E</td>
<td>E</td>
<td>C</td>
<td>68,69</td>
</tr>
<tr>
<td>Fatty foods</td>
<td>9</td>
<td>D</td>
<td>B</td>
<td>E</td>
<td>70-78</td>
</tr>
<tr>
<td>Mint</td>
<td>1</td>
<td>D</td>
<td>E</td>
<td>B</td>
<td>79</td>
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<td>Recumbent position</td>
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<td>E</td>
<td>B</td>
<td>B</td>
<td>80</td>
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<tr>
<td>RLD position</td>
<td>3</td>
<td>B</td>
<td>E</td>
<td>81-83</td>
<td></td>
</tr>
<tr>
<td>Late-evening meal</td>
<td>3</td>
<td>E</td>
<td>No effect (B)</td>
<td>B</td>
<td>84-86</td>
</tr>
</tbody>
</table>

Abbreviations: LESP, lower esophageal sphincter pressure; RLD, right lateral decubitus.

*See Table 1 for a summary of evidence A through E.
esophageal motility and gastric emptying. Modest alcohol intake has been shown to induce reflux symptoms and decrease esophageal pH in healthy individuals without GERD symptoms; despite normal overall 24-hour pH measurements. Randomized and cross-sectional studies have suggested an increased prevalence of symptomatic reflux in alcohol users. Wang et al reported reflux symptoms in 43% of heavy (≥210 g/wk) alcohol users compared with 16% of nondrinkers (OR, 2.85; 95% CI, 1.67-4.49; P = .01); however, large American and multinational cross-sectional studies have not shown similar associations.

In a case-control study (evidence B) that examined GERD outcomes in symptomatic alcoholic patients compared with matched groups (without alcohol consumption) of controls, patients with GERD, and patients with nutcracker esophagus, most alcoholic patients demonstrated LES hyperremission, high-amplitude esophageal contractions, or nonperistaltic esophageal contractions that improved after prolonged (>6 months) alcohol abstinence but were not accompanied by improvements in esophageal pH. Therefore, although esophageal motility abnormalities were shown to improve after the cessation of alcohol use, there is insufficient evidence to support the direct effect of alcohol abstinence on pH or GERD symptoms.

**OBESITY**

Obesity has been speculated to cause GERD symptoms because of multiple factors, including increased (1) gastroesophageal sphincter gradient, (2) incidence of hiatal hernia, (3) intra-abdominal pressure, and (4) output of bile and pancreatic enzymes. Although some studies have shown increased GERD in morbidly obese individuals, other studies did not demonstrate similar findings. Several population-based studies have found a significant relationship between increasing body mass index (calculated as weight in kilograms divided by the square of height in meters) and GERD symptoms. Murray et al reported an adjusted OR of 1.82 (95% CI, 1.33-2.5) for overweight patients with weekly heartburn symptoms compared with 1.3 (95% CI, 1.13-1.99) for individuals of average weight. Cross-sectional studies have similarly reported higher weekly reflux symptoms in obese patients. Another study showed that for every increment of body mass index of 5, the risk of GERD increased by 1.2. In a multivariate logistic regression model, women with a body mass index greater than 35 were shown to have an increased risk of GERD (OR, 6.3; 95% CI, 2.4-4.7) compared with men with a similar body mass index (OR, 3.3; 95% CI, 2.4-4.7; P < .001). Although some studies have reported a link between obesity and GERD by demonstrating abnormal pH and manometric measures, other studies have refuted such an association.

Bariatric surgery has been associated with improved GERD variables. Given that the surgical procedure has inherent antireflux properties, it is difficult to directly assess the effect of weight loss on GERD. Five studies have evaluated the independent effect of weight loss on GERD variables. Fraser-Moodie et al showed a significant correlation between weight loss and esophageal pH (OR, 0.55; P < .001) in an uncontrolled study of 34 obese patients with GERD. Mathus-Vliegen et al demonstrated a similar correlation with a decreased upright pH less than 4 (8.0% vs 5.5%; P < .05) and postprandial reflux episodes (49.0 vs 32.1; P < .05) in patients with a mean weight loss of 12.4 kg in 13 weeks. However, in another study that randomized 20 obese patients with reflux esophagitis to either a 430-kcal/d diet or an unrestricted diet, there was no significant difference in reflux symptoms between controls and patients with a 10% body weight loss after 6 months. Therefore, although weight loss seems to have a promising effect on pH measures and symptoms, further randomized controlled studies are warranted to determine its exact effect on GERD outcomes.

**CITRUS FRUITS AND JUICES**

Citrus fruits are often cited as precipitants of heartburn symptoms. In a questionnaire of approximately 400 patients with GERD, 72% of respondents reported increased heartburn with ingestion of either orange or grapefruit juice. However, Price et al found that acid-sensitive patients (positive Bernstein test results) were sensitive to intraesophageal infusion of orange juice, even when the orange juice was adjusted to a pH of 7, suggesting that acidity is not the only factor determining the citrus effect on GERD. Cranley et al demonstrated that patients with GERD did not change their LESP when challenged with orange juice infusion; this was in contrast to controls who showed increased LESP. No stud-

**Table 3. Summary of Lifestyle Intervention or Cessation Trials**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Trials, No.</th>
<th>Raised LESP</th>
<th>Improved pH</th>
<th>Improved Symptoms</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco</td>
<td>3</td>
<td>E</td>
<td>No effect</td>
<td>B</td>
<td>15,87,88</td>
</tr>
<tr>
<td>Alcohol</td>
<td>1</td>
<td>No effect</td>
<td>(B)</td>
<td>No effect</td>
<td>27</td>
</tr>
<tr>
<td>Weight loss</td>
<td>5</td>
<td>E</td>
<td>B</td>
<td>B</td>
<td>99-93</td>
</tr>
<tr>
<td>Coffee and caffeine</td>
<td>0</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>NA</td>
</tr>
<tr>
<td>Chocolate</td>
<td>0</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>NA</td>
</tr>
<tr>
<td>Spicy foods</td>
<td>0</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>NA</td>
</tr>
<tr>
<td>Citrus</td>
<td>0</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>NA</td>
</tr>
<tr>
<td>Carbonated beverages</td>
<td>0</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>NA</td>
</tr>
<tr>
<td>Fatty foods</td>
<td>0</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>NA</td>
</tr>
<tr>
<td>Mint</td>
<td>0</td>
<td>E</td>
<td>E</td>
<td>E</td>
<td>NA</td>
</tr>
<tr>
<td>Elevation of the HOB</td>
<td>3</td>
<td>E</td>
<td>A</td>
<td>B</td>
<td>80,94,95</td>
</tr>
<tr>
<td>LLD position</td>
<td>3</td>
<td>B</td>
<td>B</td>
<td>E</td>
<td>81-83</td>
</tr>
<tr>
<td>Late-evening meal</td>
<td>2</td>
<td>E</td>
<td>B</td>
<td>E</td>
<td>84,85</td>
</tr>
</tbody>
</table>

Abbreviations: HOB, head of the bed; LESP, lower esophageal sphincter pressure; LLD, left lateral decubitus; NA, not applicable.

*See Table 1 for a summary of evidence A through E.
ies, to our knowledge, have assessed the effect of avoidance of citrus on GERD.

**CARBONATED BEVERAGES**

Studies have postulated that carbonated beverages cause GERD. In one study, 68 carbonated soft drink consumption was found to be a predictor of GERD symptoms in a multivariate analysis. Another small study 69 of healthy individuals found equivalent decreases in LESP with ingestion of carbonated water, caffeinated cola, or caffeine-free cola compared with water ingestion. No cessation trials have been published to date, to our knowledge.

**COFFEE AND CAFFEINE**

Despite the fact that an intraesophageal infusion of coffee in patients with acid sensitivity has been shown to cause heartburn, 62 large epidemiologic studies 16,17 found no association between coffee drinking and GERD. A recent survey in Norway described a negative association between coffee drinking and GERD incidence. Although some studies 53,54 have described increased LESP with regular and decaffeinated coffees, another trial 63 showed no effect of coffee on the number of reflux episodes, total reflux time, or LESP in patients with GERD or controls. There is conflicting evidence regarding how different coffee preparations, roasting methods, and processing methods affect GERD variables. 56-59 Decaffeinated compared with caffeinated coffee ingestion was associated with significantly less acid exposure time in some studies. 60 Salmon et al 61 noted that patients had lower postprandial LESP regardless of whether they were exposed to coffee, suggesting that the association between coffee and GERD may be confounded by the fact that people frequently drink coffee after meals. Another study 62 reported that coffee decreased LESP in healthy individuals and in patients with reflux esophagitis when fasting or following a standard test meal. Given the conflicting reported data, the relationship between caffeine or coffee and GERD remains unclear. There is insufficient evidence to support the routine recommendation that patients with GERD avoid such beverages.

**CHOCOLATE**

Chocolate is frequently implicated as a provoker of GERD. There are limited data that chocolate may affect esophageal pH and LES profiles. One early uncontrolled study 63 found that ingestion of 120 mL of chocolate syrup by 9 controls caused LESP to significantly decrease compared with baseline measurements (P < .01). In 7 patients with typical GERD symptoms, Murphy and Castell 64 found greater acid exposure time after chocolate ingestion compared with ingestion of a test drink of equivalent osmolality and caloric value (P = .04). No studies have addressed the effect of chocolate abstinence on GERD symptoms.

**SPICY FOODS**

Although patients frequently cite spicy foods as an inciting agent in heartburn episodes, little data have been published regarding the effects of spicy food consumption on LESP or esophageal pH. Nebel et al 65 conducted a survey of patients with GERD symptoms and found that 88% listed spicy foods as a precipitant of heartburn. One study 66 found that in patients with GERD vs controls, onion intake increased the number of reflux episodes (P < .01) and the esophageal acid exposure time (P < .05). The results of this study have not been confirmed by other larger trials. Many patients who believe that spicy foods exacerbate their GERD symptoms may abstain from these foods although we found no published trials that studied the effect of abstinence on change in GERD symptoms.

**FATTY FOODS**

It has been postulated that fat increases not only reflux but also the sensitivity of the esophagus to acid exposure. In a randomized clinical trial, 70 8 patients with GERD and 11 controls received either an intraduodenal infusion of a lipid solution or isotonic sodium chloride. Fat infusions have not been shown to affect LESP or the rate of transient LES relaxations. 72 Nebel and Castell 73 evaluated the LES response to ingestion of fat and protein meals with equivalent caloric value in healthy individuals and found that fatty meals decreased LESP pressure significantly compared with protein meals, which increased LESP. Becker et al 74 studied esophageal acid exposure after high- and low-fat meals and found that the fat content was not significantly associated with esophageal pH abnormalities. In a study 75 of 20 healthy individuals in the supine position, those who consumed high-fat meals had significantly increased acid exposure compared with the low-fat group, but this study also found that acid exposure increased in patients who ingested a greater volume. Other randomized trials have found no difference comparing high- and low-fat meals with respect to LESP, transient LES relaxations, 76 number of reflux episodes, or esophageal exposure to acid. 77 There is conflicting evidence regarding whether caloric content of foods is more important than fat content. 76,78

Based on the previously mentioned trials, there is limited evidence to suggest that the fat content of meals affects GERD outcomes. In fact, a recent review 79 on this subject concluded that present evidence does not support any recommendations for patients with GERD to limit fat in their diets.

**MINT**

Peppermint and spearmint are plant extracts that are commonly used in foods and flavorings of toothpaste, mouthwash, and candy. Mint is commonly thought to relax the LES, and thus it is commonly recommended that patients with GERD avoided.
avoid ingesting mint.115 We found only 1 study examining the effect of mint ingestion on GERD. Bulat et al conducted a randomized double-blind placebo-controlled trial in which the LES tone of healthy volunteers was measured in response to spearmint, and they found no difference in symptoms, sphincter pressure, or reflux episodes with ingestion of spearmint. There were no studies published on the effect of cessation of mint intake on GERD symptoms.

LATE-EVENING MEAL

Postprandial reflux is common in patients with GERD.116 Two studies that evaluated the effect of the timing of the evening meal on 24-hour intragastric acidity in healthy volunteers showed different effects on nocturnal pH. A study of early (6 PM) vs late (9 PM) dinner demonstrated lower intragastric pH (median pH, 1.39 vs 1.67, P < .001) in the later meal setting, but only between midnight and 7 AM. The second study in 10 healthy patients showed that nocturnal and 24-hour integrated intragastric acidity were unaffected by changes in the timing of the evening meal. In the only study in patients with GERD that examined the effect of late-evening meals, Orr and Harinish measured esophageal pH and symptoms in 20 patients during a late-evening meal on one night and a meal before 7 PM on another night. There was no difference between the 2 nights in terms of acid exposure, number of reflux episodes, and mean reflux episode duration.80 The evidence to routinely recommend avoidance of late-evening meals in patients with GERD remains incomplete.

RECUMBENT POSITION AND HOB ELEVATION

Counseling patients regarding HOB elevation is based on the theory that stomach contents containing acid will more likely reflux into the esophagus while patients are lying flat. Stanciu and Bennett compared the effect of different body positions (sitting, lying, and elevated HOB) in 63 patients with GERD symptoms using pH monitoring and showed that compared with patients who slept flat, patients who slept with an elevated HOB (using 28-cm blocks) had significantly fewer reflux episodes, shorter reflux episodes, faster acid clearing, and fewer reflux symptoms. Hamilton et al conducted a randomized clinical trial comparing sleeping on a wedge, sleep with HOB elevation, and sleeping in the horizontal position and found that sleeping on a wedge was associated with significantly less esophageal acid exposure compared with sleeping in the horizontal position. Sleeping with HOB elevation also showed improved acid exposure, but this difference was not statistically significant.91 In another randomized controlled trial of HOB elevation for 2 weeks in patients with reflux symptoms treated with proton pump inhibitors and cisapride, HOB elevation was not associated with a difference in symptoms or antacid use. The HOB elevation, therefore, seems to be an effective measure to improve the symptoms and physiologic variables in some patients with GERD.

RIGHT LATERAL DECUBITUS POSITION

Several studies have shown that reflux is increased in patients in the right lateral decubitus position. The reason for this phenomenon is not completely clear, but it may be related to increased transient LES relaxations in the right position, or possibly that the gastroesophageal junction lies above the level of gastric acid in the left lateral position.113 Specifically, total reflux time, average acid clearance, and LES relaxations are significantly prolonged in patients lying on their right side compared with the left lateral decubitus position.81-83 Despite evidence that the right lateral decubitus position can aggravate reflux, the practical aspects of this lifestyle modification make it a challenge to implement in patients with GERD.

CONCLUSIONS

Despite articles in the literature emphasizing the insufficient evidence to support an association between lifestyle and dietary behaviors and GERD,115,117 such interventions continue to be recommended as first-line therapy.1,118 Gastroesophageal reflux disease is a chronic disease that affects health-related quality of life.119 The results of this evidence-based approach suggest that although there is physiologic evidence that smoking, alcohol, chocolate, or fatty or citrus food intake may adversely affect symptoms or esophageal pH, there is little evidence that cessation of these agents will improve GERD variables. Elevations in the HOB, left lateral decubitus positioning, and weight loss have been associated with improvement in GERD variables in case-control studies. Larger prospective controlled trials are warranted to conclusively recommend dietary and lifestyle modifications in the treatment of GERD.

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