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EFFECTS OF GUM CHEWING ON PHARYNGEAL AND ESOPHAGEAL PH

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We investigated the effects of gum chewing on pharyngeal and esophageal pH levels in patients with laryngopharyngeal reflux (LPR) who were undergoing reflux testing. Forty consecutive, unselected, adult patients who were undergoing ambulatory double-probe (simultaneous pharyngeal and esophageal) pH monitoring for diagnosis of LPR were asked to chew 2 sticks of gum 4 times during their pH studies. Twenty subjects chewed regular sugarless gum, and 20 subjects chewed a sugarless gum containing bicarbonate. The subjects recorded the beginning and end of each gum-chewing period. The mean pH values for the gum-chewing intervals and for comparable pre–gum-chewing intervals were analyzed statistically for both the pharyngeal and esophageal probe data. The regular gum group and the bicarbonate gum group were analyzed separately. In addition, the gum-chewing pH data were compared to controls, ie, normal postcibal buffering effects. The data show that gum chewing consistently increases esophageal and pharyngeal pH, and that bicarbonate gum causes greater increases than regular gum. For patients with LPR, gum chewing appears to be a useful adjunctive antireflux therapy.

KEY WORDS — bicarbonate, chewing gum, esophagus, gastroesophageal reflux, GERD, laryngopharyngeal reflux, pH monitoring, reflux, reflux treatment.

INTRODUCTION

Laryngopharyngeal reflux (LPR) is distinctly different from classic gastroesophageal reflux disease (GERD).[1] The hallmark of GERD is esophagitis and its principal symptom, heartburn. Patients with LPR have dysphonia, chronic throat clearing, globus, and dysphagia as the primary symptoms, and usually do not have heartburn (or esophagitis).[1] The differences between LPR and GERD may be explained in part by differences in the patterns and mechanisms of reflux in each group.[2] Typically, GERD patients have supine (nocturnal) reflux (with long periods of acid and peptic exposure in the esophagus). By comparison, LPR patients usually have daytime (upright) reflux with normal esophageal function and minimal nighttime reflux.[1,2] A certain amount of reflux is normal during esophageal pH studies, but so-called "physiologic reflux" in the pharynx is never considered normal.[1,2] In many LPR patients, postcibal symptoms persist despite aggressive antireflux treatment.

Unlike the esophagus, which secretes bicarbonate,[3] the laryngopharyngeal mucosa lacks this intrinsic protection against the effects of reflux. The larynx also lacks significant extrinsic sources of protection. In the esophagus, peristalsis and salivary bicarbonate provide for restoration of neutral pH after a reflux episode has occurred.[4] The laryngopharynx has no such acid clearance mechanism.

Gum chewing has been shown to increase salivary flow rates, salivary bicarbonate concentration, and the rate of swallowing.[5-7] In addition, increases in salivary flow appear to be a function of gum-stick size, ie, the larger the bolus of gum, the greater the increase in salivary flow.[7,8] Also, the specific beneficial effects of sugar-free gum on the mouth and esophagus have been reported.[5-8] We investigated the effects of gum chewing on esophageal and pharyngeal pH in patients who were undergoing ambulatory 24-hour double-probe (simultaneous esophageal and pharyngeal) pH monitoring.

MATERIALS AND METHODS

Forty consecutive, unselected adult patients who were undergoing ambulatory double-probe pH monitoring for evaluation of LPR were studied prospectively. The subjects were either not on antireflux therapy, or had been asked to stop therapy 1 week before testing, as is our routine for reflux testing. The protocol for this study was approved by the Wake Forest University School of Medicine institutional review board for human research.

The first 20 subjects were given 8 sticks of Wrigley's Winterfresh Sugarfree Gum and instructed to chew 2 sticks at a time. The subjects were instructed to chew for a minimum of 10 minutes and allowed to chew as long as they wished, and they were instructed to use an event marker to indicate the gum-chewing periods. Similarly, the second 20 patients were given 8 sticks of Arm and Hammer Sugarfree

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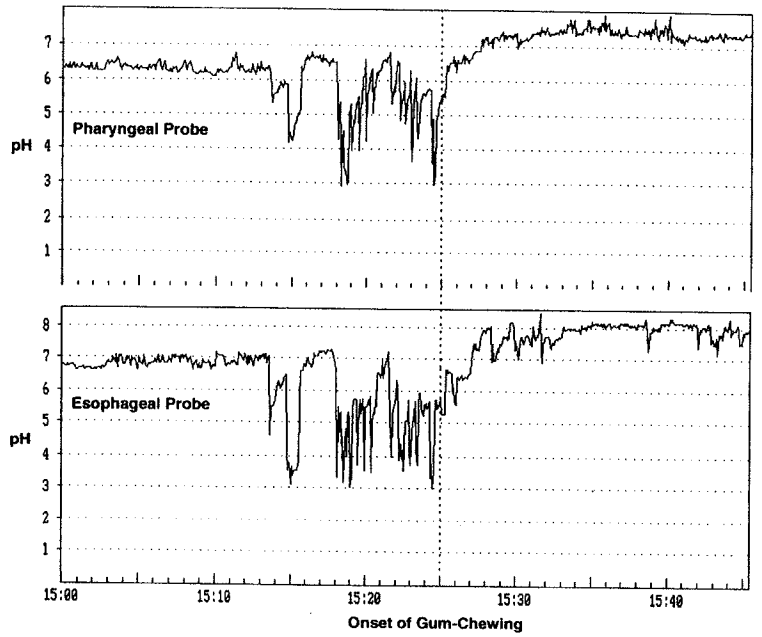

pH tracing demonstrates effects of gum chewing. Shown here is portion of double-probe (simultaneous esophageal and pharyngeal) pH tracing of study subject. Before gum chewing, subject demonstrated multiple esophageal and pharyngeal reflux episodes (to below pH 4), and he was symptomatic. Shortly after he began to chew bicarbonate gum, pH was restored to neutral (normal) in both esophagus and pharynx. In instance shown, disappearance of patient's symptoms coincided with gum chewing and with observed pH shifts.

Baking Soda Gum.

After completion of each study, the data were analyzed. The mean pH of each gum-chewing period was compared to that of the comparable time period just prior to gum chewing. The first 5 postcibal minutes were excluded, as it has been demonstrated that physiologic clearing of gastric contents may occur during that time. The data were analyzed by Student's *t*-test for both the pharyngeal and esophageal probes for both types of chewing gum. Similar statistical analysis was applied to the differences between the effects of gum chewing and control data.

For a long time, frequent small meals have been recommended for patients with reflux. Presumably, this recommendation was based upon the observation that small meals are less likely to cause reflux, but equally important, the antireflux effects of eating also are presumed to be related to the buffering effects of food on the stomach contents. Thus, the question that we attempted to address using controls was, Does food provide the same degree of buffering and postcibal symptom relief as does gum chewing?

Ten (other) patients with clinical LPR who were undergoing pH monitoring were used as the controls. The pH recordings of the control subjects were analyzed to determine the mean duration of the buffering effect of food (after completing a meal); ie, we measured the time from the end of each meal to the first postcibal reflux episode.

RESULTS

The regular gum (RG) and the bicarbonate gum (BG) groups were analyzed separately. In 92% (37/40) of the subjects, the mean esophageal and pharyngeal pH increased with gum chewing. For the RG group, the mean pharyngeal pH increased from 6.85 to 7.30 during the gum-chewing period ($p < .0001$), and the mean esophageal pH increased from 6.45 to 7.14 ($p < .0001$). For the BG group, the mean pharyngeal pH increased from 6.70 to 7.43 during gum chewing ($p < .0001$), and the mean esophageal pH increased from 6.44 to 7.49 ($p < .0001$). In some subjects, gum chewing appeared to completely abolish reflux events (see Figure).

The mean (±SD) time of gum-chewing episodes was 25.2 ± 16.0 minutes, and the mean buffering effect of BG chewing was 49.1 ± 23.6 minutes. This difference was highly significant at the $p < .0001$ level. On average, the beneficial effect of gum chewing on pH was maintained 2.4 ± 1.3 times as long as the gum-chewing period (range, 1.1 to 7.0 times), and in no case did reflux occur during gum chewing.

The control data revealed that the mean buffering effect of meals — the time from the end of the meal to the first postcibal reflux episode — was 27.4 ± 18.2 minutes. The difference between the mean duration of the gum-chewing effect and the mean duration of the postcibal buffering effect (controls) was also statistically significant ($p = .002$).

DISCUSSION

On the basis of anecdotal reports of LPR patients who experienced relief of LPR symptoms (particularly postcibal symptoms) after gum chewing, a literature review was performed. It revealed that signif-

icant increases in salivary flow and salivary bicarbonate concentration are associated with gum chewing, and that chewing gum appears to be therapeutic for GERD.[6-8] The study herein reported is the first aimed at assessing the potential benefits of gum chewing on reflux in LPR patients.

Esophageal acid clearance is a combination of peristalsis (volume clearance of the acid bolus) and the subsequent delivery of salivary bicarbonate.[4] After an esophageal reflux event, even after the acid bolus has been cleared (by a swallow), the intraluminal pH remains low. Restoration of esophageal pH to neutral levels occurs as a result of the delivery of salivary bicarbonate by subsequent swallows. Although gum chewing may also increase swallowing rates and peristalsis, this hypothesis has not been studied.

Our data indicate that substantial and significant pH shifts follow gum chewing in most subjects. On average, the beneficial (buffering) effects of chewing BG last more than twice as long as the actual gum-chewing periods, and the beneficial effects appear to be significantly longer than the buffering effects of ordinary meals (49 minutes versus 27 minutes; $p = .002$). These data are consistent with our clinical experience that BG gum chewing can provide dramatic symptomatic relief for LPR patients, especially those who have symptoms after meals.

We currently recommend that patients with postcibal symptoms chew 2 pieces of BG one half-hour after a meal or at the time of onset of symptoms, whichever comes first. In addition, we recommend gum chewing for LPR patients at certain other times, depending on symptom patterns, eg, before (or after) exercise, before going to bed, etc. Although the effect is difficult to quantify, chewing BG appears to be an inexpensive, well-tolerated, and effective adjunct to traditional drug therapy for LPR.

CONCLUSIONS

Our data demonstrate that gum chewing consistently increases both esophageal and pharyngeal pH, and that in LPR patients, BG chewing appears to be a useful adjunctive antireflux therapy.

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REFERENCES

1. Koufman JA. The otolaryngologic manifestations of gastroesophageal reflux disease (GERD): a clinical investigation of 225 patients using ambulatory 24-hour pH monitoring and an experimental investigation of the role of acid and pepsin in the development of laryngeal injury. Laryngoscope 1991;101: (suppl 53).

2. Koufman JA, Sataloff RT, Toohill R. Laryngopharyngeal reflux: consensus conference report. J Voice 1996;10:215-6.

3. Orlando RC. Esophageal epithelial resistance. J Clin Gastroenterol 1986;8(suppl 1):12-6.

4. Helm JF. Esophageal acid clearance. J Clin Gastroenterol 1986;8(suppl 1):5-11.

5. Jensen ME, Wefel JS. Human plaque pH responses to meals and the effects of chewing gum. Br Dent J 1989;167:204-8.

6. Yankell SL, Emling RC. Clinical study to evaluate the effects of three marketed sugarless chewing gum products on plaque pH, pCa, and swallowing rates. J Clin Dent 1989;1:70-4.

7. Rosenhek M, Macpherson MD, Dawes C. The effects of chewing-gum stick size and duration of chewing on salivary flow rate and sucrose and bicarbonate concentrations. Arch Oral Biol 1993;38:885-91.

8. von Schonfeld J, Hector M, Evans DF, Wingate DL. Oesophageal acid and salivary secretion: is chewing gum a treatment option for gastro-oesophageal reflux? Digestion 1997;58: 111-4.