GASTROENTEROLOGY 1988;95:581–8

ALIMENTARY TRACT

Double-Blind Controlled Trial of the Garren–Edwards Gastric Bubble: An Adjunctive Treatment for Exogenous Obesity

STANLEY B. BENJAMIN, KATHLEEN A. MAHER, EDWARD L. CATTAU, Jr., MARTIN J. COLLEN, DAVID E. FLEISCHER, JAMES H. LEWIS, CECELIA A. CIARLEGLIO, JERRY M. EARLL, SUZANNE SCHAFFER, KENNETH MIRKIN, JAMES COOPER, and AARON M. ALTSCHUL

Gastroenterology Division, Department of Medicine and Eating Disorders Clinic, Georgetown University Hospital, Washington, D.C.; and Gastroenterology Division, Fairfax Hospital, Fairfax, Virginia

Since its approval by the Food and Drug Administration in September 1985, the Garren–Edwards gastric bubble has been extensively used as an adjunct to diet and behavioral modification in the treatment of exogenous obesity. In an attempt to evaluate the efficacy of the Garren–Edwards gastric bubble, a double-blind crossover study was undertaken. Ninety patients were randomized into three groups: bubble-sham, sham-bubble, and bubble-bubble in two successive 12-wk periods. Sixty-one patients completed the entire 24-wk study. All groups participated in ongoing diet and behavioral modification therapy in a free-standing obesity program, the members of which were blinded to randomization arms. All patient groups lost weight during this study. The mean cumulative weight loss in pounds at 12 wk was as follows: bubble-sham = 19, sham-bubble = 12, and bubble-bubble = 8; and at 24 wk: bubble-sham = 23, sham-bubble = 16, and bubble-bubble = 18. The mean cumulative change in body mass index (kg/m^2) at 12 wk was as follows: bubble-sham = −3.1, sham-bubble = −2.3, and bubble-bubble = −2.9; and at 24 wk: bubble-sham = −3.1, sham-bubble = −3.0, and bubble-bubble = −3.3. Although weight loss occurred more consistently in patients with a Garren–Edwards gastric bubble, there were no significant differences between any of the three groups at 12 or 24 wk with respect to weight loss or change in body mass index. The major part of the weight loss noted during this study occurred during the first 12-wk period, irrespective of therapy (bubble or sham). Side effects observed during this study included gastric erosions (26%), gastric ulcers (14%), small bowel obstruction (2%), Mallory–Weiss tears (11%), and esophageal laceration (1%). We conclude that, in this study, the use of a Garren–Edwards gastric bubble did not result in significantly more weight loss than diet and behavioral modification alone in the management of exogenous obesity, and it may result in significant morbidity.

Obesity represents a significant health hazard in this country. Approximately 30% of men and 20% of women are currently considered to be obese, and 5% of men and 7% of women are morbidly obese and have severe ongoing medical problems as a direct consequence of their obesity (1–3). This degree of obesity has been shown in population studies to be associated with increased risks for coronary artery disease, hypertension, stroke, cholelithiasis, diabetes mellitus, osteoarthritis, pulmonary embolism, gastroesophageal reflux, and psychosocial dysfunction (1–3).

Given the scope of the problem and its attendant morbidity, a large number of diverse methods to achieve weight loss have been introduced. Although diet, the simplest method, is effective, the requirement for strict adherence and long-term compliance

Abbreviations used in this paper: BMI, body mass index; C, compliant; EDC, Eating Disorder Clinic; GE, gastric erosion; GEGB, Garren–Edwards gastric bubble, GU, gastric ulcer; NC, noncompliant; SBO, small bowel obstruction.

0016-5085/88/$3.50

has been associated with an extremely high failure rate (4,5). Surgical methods have also been used, but many individuals who are significantly overweight do not qualify as operative candidates because they are not morbidly obese and, moreover, the surgery itself is associated with significant morbidity and alterations in normal physiologic function (6,7).

Clinicians have long recognized that one of the major signs of naturally occurring or postgastric surgery bezoars is weight loss (8). The exact reason has never been completely understood, but it is assumed that a decrease in available gastric volume results in diminished gastric capacity, promoting early satiety. Based on this observation, there was a series of uncontrolled reports in which weight loss was achieved by implanting a variety of devices in the stomach, such as silicone breast implants and toy balloons (9–12).

Experiments using an artificial bezoar for the treatment of obesity were initially reported in the United States by Garren and Garren (12–14) using a cylindrical polyurethane device designed to be placed and removed endoscopically. Despite encouraging data submitted before its approval in September 1985, there was no available, controlled information on the efficacy of this device in promoting weight loss beyond that achieved by diet and behavioral modification alone. Therefore, in February 1986, we undertook a study to determine the efficacy of the Garren–Edwards gastric bubble (GEGB) as an adjunctive therapy in the treatment of exogenous obesity.

Materials and Methods

Patients

All patients expressing interest in the GEGB as a means of achieving weight loss were referred to the Eating Disorders Clinic (EDC) at Georgetown University Hospital, Washington, D.C., a free-standing clinic specializing for many years in the treatment of obesity with diet and behavioral modification. Patients were independently evaluated by an internist, a dietician, and a psychologist. They were required to be at least 30% above ideal body weight and to have previously failed to lose weight in an organized weight loss program. Physical examination and psychologic screening were performed and dietary history was obtained. Prospective candidates were reviewed by members of the EDC, and only those patients with no medical or psychologic problems and who agreed to return for follow-up and continuous treatment at the EDC were considered eligible for randomization.

Patients were not considered eligible for randomization if they were deemed psychologically or physically unable to maintain regular follow-up at the EDC or if they had medical problems precluding safe endoscopy. Patients requiring continuous therapy with nonsteroidal antiinflammatory drugs, corticosteroids, or anticoagulants were also excluded. Other reasons for exclusion included active ulcer disease, previous complications of ulcer disease, previous intestinal surgery, structural abnormalities of the gastrointestinal tract, lesions considered at risk for bleeding (e.g., polyps, arteriovenous malformations, tumors), and pregnancy.

Study Outline

A graphic representation of the study is given in Figure 1. After approval by the EDC, patients were referred to the Gastroenterology Clinic where written, informed consent was obtained. Endoscopy was performed to clear the upper gastrointestinal tract of any lesion precluding GEGB insertion. This occurred coincidentally with obtaining measurements necessary for safe passage of the GEGB (see below). At this point, randomization was done by sealed envelope. Patients were randomly assigned to one of three groups: sham-bubble (SB), bubble-sham (BS), or bubble-bubble (BB). After 12 wk of concurrent diet and behavioral modification therapy in the EDC and biweekly follow-up by a nurse practitioner in the Gastroenterology Clinic to assess any symptoms possibly related to the GEGB, patients again underwent endoscopy. The GEGB was removed (if present) and replaced for SB and BB patients. After a second 12-wk period, all patients again underwent endoscopy, the GEGB was removed (if present), and the patients were exited from the study. This protocol was approved by the Institutional Review Board at Georgetown University. Patients were not charged for the GEGB or the endoscopic procedures.

Endoscopic Techniques

All endoscopies were performed by fully trained staff endoscopists actively involved in a wide array of therapeutic procedures who had been specially trained in the use of the GEGB. The GEGBs were provided at no charge by the manufacturer, American-Edwards Laboratories, Inc., Santa Ana, Calif. Before each insertion, all devices were tested for leaks by placing a fully inflated bubble under water. The bubble was then loaded into the introducer tube by one of two nurses trained in the procedure as outlined in the manufacturer's instructions. Patients were premedicated with meperidine and either diazepam or midazolam. A towel was used to cover the patient's eyes, further ensuring lack of knowledge of GEGB

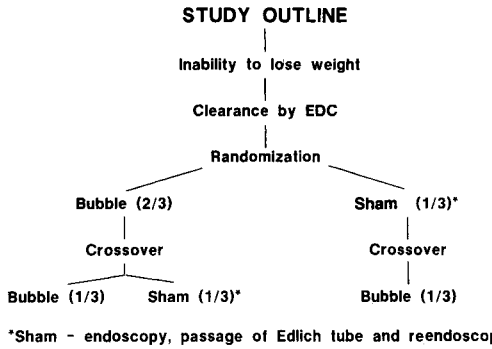

Figure 1. Study outline for the Garren–Edwards Gastric Bubble Study.

placement or sham. Using a pediatic endoscope (GIFP or GIFXP; Olympus Corporation, Lake Success, N.Y.) diagnostic endoscopy was performed. This was a complete esophagogastroduodenoscopy with examination extended to the postbulbar duodenum. During withdrawal of the endoscope, the distance from the incisor teeth to the diaphragmatic hiatus was measured and recorded.

After removal of the endoscope, the GEGB introducer was passed to a level at least 5 cm beyond the measured diaphragmatic hiatus. If resistance was encountered during insertion, a pediatric scope was passed alongside the introducer or placed before insertion of the introducer to ensure proper positioning. The GEGB was deployed by inflation with 200 ml of room air. The inflation cannula was pulled out of the introducer tube and then the introducer tube was removed. The endoscope was reintroduced to ensure proper bubble inflation and deployment and to assess any injury to the gastrointestinal tract from either the introducer tube or the device. Sham procedures were carried out in an identical manner except that an empty Edlich tube (Monoject, St. Louis, Mo.), which approximated the GEGB introducer tube in size and consistency, was passed to simulate passage of the GEGB introducer tube. A repeat endoscopy was then performed.

At the time of midtrial crossover or exit from the study, the endoscopy was performed using the Olympus therapeutic endoscope (GIF2T10). Removal of the GEGB was accomplished by puncturing the device using either a sclerotherapy needle, a thermal device (heater probe, Bicap tumor probe, or laser), or endoscopic scissors. The GEGB was then firmly grasped and removed with rat-toothed forceps, a polypectomy snare, or a coaxial balloon. A repeat panendoscopy with a pediatric endoscope was always performed to evaluate mucosal injury, either from the GEGB or as a consequence of its removal.

Follow-up

Patients were regularly followed in both the EDC and the Gastroenterology Clinic. Patients were encouraged to attend weekly classes in the EDC that addressed concepts of diet adherence and behavioral modification in an attempt to relearn proper eating habits. Members of the EDC were not aware of the treatment arm to which any of the patients had been randomized. Attendance records of these visits were kept. Patients were also seen biweekly in the Gastroenterology Clinic by a nurse practitioner who was blinded to the patient's randomization. Patients were weighed and questioned about any symptoms they experienced, and they filled out a questionnaire asking if they thought that a GEGB was in place.

All patients were given similar instructions following entry into the protocol. They were placed on a liquid diet (800 cal/day) for 1 wk and were subsequently given an individualized caloric intake schedule in a manner standard for the EDC. All patients were instructed to take antacids (30 ml orally after meals and at bedtime) and dicyclamine (Bentyl Merrell Dow Pharmaceuticals, Cincinnati, Ohio; 30 mg orally every 4–6 h as required) for abdominal cramps. No H_2-blockers were given. All patients were given verbal and written instructions to contact the Gastroenterology Clinic for persistent abdominal pain, nausea, vomiting, or diarrhea. Patients with these complaints were asked to report to the clinic and were evaluated by a physician who was not involved with their previous endoscopy. Decisions regarding the appropriate clinical evaluation of these symptoms were made by physicians unaware of the patient's randomization status. Patients with persistent symptoms underwent endoscopy to exclude ulcers as the cause and to assess the status of the bubble, if present. If an ulcer was seen, the GEGB was removed. If no bubble was present, the record was reviewed to ascertain their randomization status. If a bubble had been placed but was not found, a complete evaluation was undertaken (physical examination, complete blood count, plain abdominal films, and upper gastrointestinal radiographs with small bowel follow-through) to determine the presence or absence of the device in the gastrointestinal tract. Patients with a GEGB in the small bowel were hospitalized until the GEGB passed spontaneously or until surgery for removal was performed. Those patients in whom a GEGB had been placed but was not found by any study were assumed to have passed the device spontaneously.

Because of our concern with bubble deflation, after 12 wk all noncompliant patients (who failed to return to the Gastroenterology Clinic for scheduled endoscopy) were contacted by the Gastroenterology Service, initially by phone and then by registered mail and telegram if no response was received.

Statistical analysis was performed using Student's *t*-test or χ^2 analysis, where appropriate.

Results

From February 1986 to April 1987, 90 patients were entered into this study. There were 17 men and 73 women (mean age 38.1 yr, range 14–56 yr; mean weight 254 lb, range 162–383 lb). They were randomized into one of the three groups: BS, SB, or BB.

Table 1. Patients Who Withdrew From the Garren–Edwards Gastric Bubble Study

	Total No. of patients	Group BS	SB	BB
Requested withdrawal (no symptoms or complications)	11	4	2	5
Gastric ulcers with pain	3	1	0	2
Became pregnant during study	2	2	0	0
Developed abdominal pain, no ulcer found	1	0	0	1
Inability to tolerate crossover endoscopy	1	0	1	0
Small bowel obstruction[a]	3	0	1	2
Lost to follow-up	8	4	2	2
Total	29	11	6	12

BB, bubble-bubble; BS, bubble-sham; SB, sham-bubble. [a] Two patients had bowel obstruction caused by a gastric bubble; 1 patient with a sham bubble developed obstruction caused by adhesions.

From the original group of 90 patients, 61 completed the entire 24 wk of therapy. Twenty-nine patients withdrew from the study (Table 1). Eleven patients requested withdrawal without developing symptoms or complications; 3 had gastric ulcers with pain; 2 became pregnant; 1 patient developed abdominal pain without ulcer; 1 patient could not tolerate endoscopy at crossover; and 3 patients had small bowel obstruction, two due to deflation and passage of the device into the small bowel and one secondary to adhesions in a sham patient. Eight patients did not return for follow-up despite repeated attempts to contact them. The three patient groups completing the study were similar with respect to age, sex, initial weight, and body mass index (BMI = kg/m^2) (Table 2).

Weight loss as a reflection of the change in BMI (ΔBMI) is shown in Figure 2. All three groups lost weight during the course of the study although there was wide individual variation. The greatest change in BMI occurred during the first 12 wk of the study, irrespective of whether a bubble had been inserted. The change in BMI for each of the groups at 12 wk was as follows: BS = -3.1 kg/m^2, SB = -2.3 kg/m^2, and BB = -2.9 kg/m^2 (Table 2). There were no statistical differences between these groups (BS vs. SB, $p = 0.3$; BS vs. BB, $p = 0.6$; SB vs. BB, $p = 0.6$, Student's *t*-test.) However, all patients with bubbles lost some weight at 12 wk (BB, 15 of 15; BS, 19 of 19), but only 67% (18 of 27) of patients with sham bubbles lost weight ($p < 0.001$, χ^2 test). During the second 12-wk period, a decrease in BMI was also noted, but in all groups this was less than the original 12-wk period (Table 1, Figure 2; ΔBMI: BS = 0.0 kg/m^2, SB = -0.7 kg/m^2, BB = -0.4 kg/m^2). At the completion of the second 12-wk period, there were no statistically significant differences between the groups (BS vs. SB, $p = 0.3$; BS vs. BB, $p = 0.5$; SB vs. BB, $p = 0.6$, Student's *t*-test.) At 24 wk, 94% (32 of 36) of patients receiving a bubble in the first 12 wk had cumulative weight loss and 70% (19 of 27) of patients with a sham bubble in the first 12 wk had a cumulative weight loss ($p < 0.02$, χ^2 test).

Table 2. Study Groups at Completion of the Garren–Edwards Gastric Bubble Study[a]

	BS (n = 19)	SB (n = 27)	BB (n = 15)
Sex			
Male	5	5	0
Female	14	22	15
Age (yr)			
Mean	39.9	38.1	38.5
SD	7.9	9.6	10.8
Range	28–55	14–55	24–55
Initial weight (*lb*)			
Mean	271	254	237
SD	55	48	39
Range	222–375	177–383	162–305
BMI (kg/m^2)			
Mean	44.8	42.9	39.9
SD	7.9	6.4	6.6
Range	33.8–66.6	32.3–58.5	30.4–55.5

BB, bubble-bubble; BMI, body mass index; BS, bubble-sham; SB, sham-bubble. [a] Total number of patients = 61.

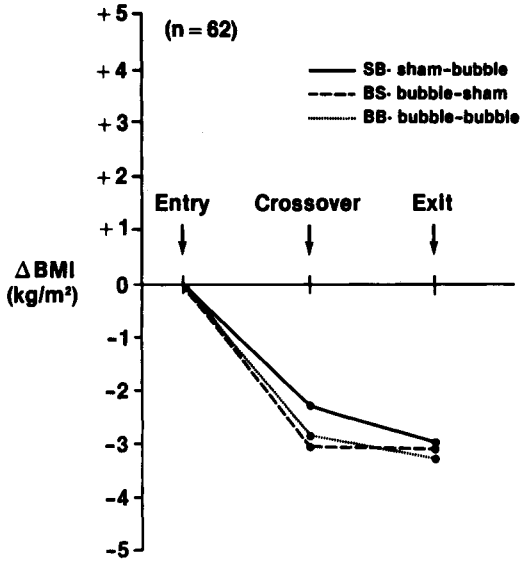

Figure 2. Change in BMI (body mass index, kg/m^2) over the course of the 24-wk study. Weight changes represent the cumulative changes over the entire period (BS, bubble-sham; SB, sham-bubble; BB, bubble-bubble).

Weight loss changes in pounds for each 2-wk interval are shown in Figure 3. Patients demonstrated the greatest absolute weight loss during the first 12-wk period irrespective of their group. Insertion of a GEGB at the 12-wk crossover did not result in accelerated weight loss in the SB or BB groups.

Patients in each group were divided into compliant (C) and noncompliant (NC) groups. A compliant patient was defined as one who attended >50% of the EDC classes. The changes in BMI for each of the groups is shown in Figure 4. All compliant patients had a greater change in BMI at the end of 12 and 24 wk than their noncompliant counterparts. However, in no group did the presence of a GEGB correct for noncompliance; the compliant sham patients lost more weight than the noncompliant GEGB patients (at 12 wk: C-SB, ΔBMI = -2.6; NC-SB, ΔBMI = -1.9; C-BS, ΔBMI = -3.8; NC-BS, ΔBMI = -1.9; C-BB, ΔBMI = -3.6; NC-BB, ΔBMI = -1.9; C-SB × NC-BS, $p = 0.13$; C-SB × NC-BB, $p = 0.11$; at 24 wk: C-SB, ΔBMI = -3.1; NC-SB, ΔBMI = -2.8; C-BS, ΔBMI = -3.7; NC-BS, ΔBMI = -1.9; C-BB, ΔBMI = -4.5; NC-BB, ΔBMI = -1.7; C-BS × NC-SB, $p = 0.20$; C-BS × NC-BB, $p = 0.06$; Student's *t*-test).

The patients' responses to the questions asked at

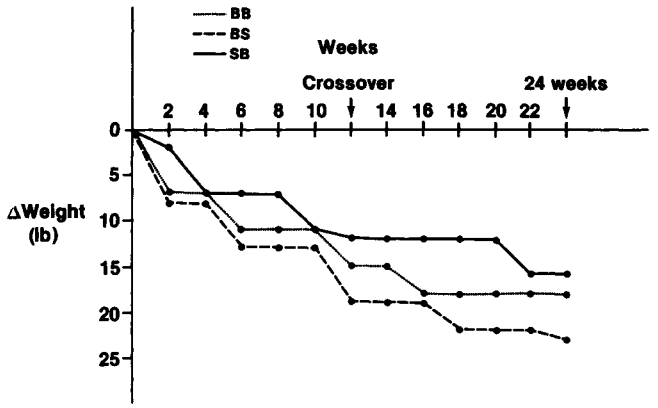

Figure 3. Cumulative weight loss over the entire 24-wk study for each of the three study groups (BS, bubble-sham; SB, sham-bubble; BB, bubble-bubble).

follow-up visits about the presence or absence of a GEGB are shown in Table 3. Only 65% of the patients with a GEGB in place answered affirmatively, 19% negatively ($p < 0.001$, χ^2 test). Forty-two percent of sham patients said they felt a GEGB was present.

Complications encountered during the trial are listed in Table 4. These can be considered as complications of the insertion of the device, those occurring as a result of having the GEGB in place, and those complications associated with GEGB removal.

Incomplete deployment, defined as an inadequately inflated device or failure of the bubble to exit the introducer tube, occurred in 3% (3 of 90) patients). This required immediate removal of the device and reinsertion of a new GEGB. Removal of these devices was considerably more difficult because of their stiffness. In 1 patient, inflation of the

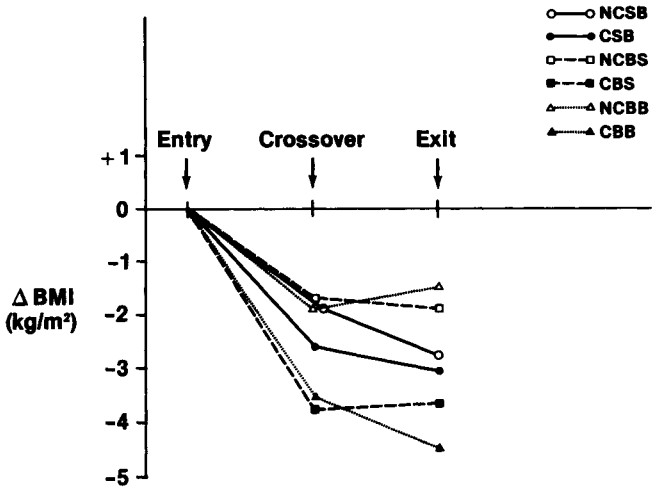

Figure 4. Change in BMI (kg/m^2) over the course of the 24-wk study for compliant (C) and noncompliant (NC) patients. NC-SB, noncompliant sham-bubble; C-SB, compliant sham-bubble; NC-BS, noncompliant bubble-sham; C-BS, compliant bubble-sham; NC-BB, noncompliant bubble-bubble; C-BB, compliant bubble-bubble.

Table 3. Patient Responses to the Question: "Is There a Bubble in Your Stomach?"

	Response (%)		
	Yes	No	Uncertain
Bubble present	65[a]	19[a]	16
Sham	42	45	15

[a] $p < 0.001$, χ^2 test.

device was associated with severe substernal pain. Inflation was stopped at 100 ml of air and a pediatric scope was passed revealing a partially inflated GEGB lodged in the distal esophagus. The device was deflated and pushed into the stomach. Examination of the esophagus revealed lacerations. The device was removed and the patient was monitored closely. After complete healing, the patient was rerandomized and a GEGB was later inserted under direct vision.

Complications noted during the time the GEGB was in place were as follows: gastric erosion (GE), gastric ulcer (GU), deflation with spontaneous passage, and deflation with small bowel obstruction (SBO) caused by impacted GEGB (Table 4). Gastric erosions were commonly seen during use of GEGB (26%, 23 of 90 patients) with most characterized as variolliform erosions, i.e., a raised mound with an erosion on top, almost uniformly found along the greater curvature of the stomach at the junction of the body and antrum. In 3 patients with GEs there were areas of white plaquelike material in the same greater curvature distribution. Pathologically these were interpreted as hyperplasia of gastric epithelium covered by an acute inflammatory infiltrate. Gastric ulcers (size range 1–3 cm) were seen in 14% (13 of

Table 4. Complications Encountered During Insertion and Removal of 90 Garren–Edwards Gastric Bubbles

	No. of patients	Percentage
Complications of insertion		
Incomplete deployment	3	3
Esophageal laceration	1	1
Complications of GEGB in place		
Gastric erosions[a]	23	26
Gastric ulcer	13	14
Deflation, spontaneous passage	6	7
Deflation, small bowel obstruction[b]	2	2
Complications during removal		
Mallory–Weiss tear	10	11
Inability to remove device	0	0
Respiratory compromise	0	0

GEGB, Garren–Edwards gastric bubble. [a] "Hyperplastic" gastritis seen in 3 patients. [b] In addition to the 2 patients with small bowel obstruction due to GEGB, 1 sham patient required operation for small bowel obstruction.

90) of patients during the time the GEGB was in place. No patient in the sham group developed GUs or GEs. These ulcers were found along the greater curvature of the stomach at the junction of the body and antrum, often with the front edge of the GEGB lying in the ulcer crater. In 6 of 13 of these patients no symptoms referable to the ulcer were reported. In the remaining 7 patients, symptoms of varying severity were recorded. Three patients exited the study because of severe pain secondary to GUs.

Small bowel obstruction caused by spontaneous deflation of the bubble, which then passed into and impacted the small bowel, occurred in 2 patients. In 6 other patients, the GEGB deflated but passed uneventfully through the gastrointestinal tract. In the 2 patients with SBO, the acute onset of abdominal pain, nausea, vomiting, and diarrhea led to evaluation disclosing the absence of a GEGB in the stomach with subsequent development of clinical SBO. Both patients were hospitalized; 1 patient responded to conservative medical management with eventual spontaneous passage of the GEGB, the other patient required surgical exploration with enterotomy for GEGB removal. Both of these patients had been noncompliant and failed to return at 12 wk for crossover or exit, despite repeated attempts (phone calls and registered mail) to contact them. One additional patient during the sham arm of the study presented with SBO and underwent exploratory laparotomy. Adhesions from previous surgery were discovered as the cause.

The most frequent complication noted during removal of the GEGB was Mallory–Weiss tears secondary to trauma at the cardioesophageal junction or retching associated with the procedure, or both. Mallory–Weiss tears were seen in 11% (10 of 90) of patients. However, none of these patients required specific therapy as all bleeding was minimal and stopped spontaneously.

In several patients, the GEGB slipped off the grasping forceps or snare, or the material tore, allowing the device to fall back into the stomach from the esophagogastric junction. In these instances, a second and, occasionally, third attempt was needed, but it was always successful. In no patient did the bubble become impacted in the pharynx, and no respiratory problems were encountered with removal.

Discussion

The magnitude of the problem of exogenous obesity, with its attendant medical morbidity and mortality along with its social implications, has fostered a wide variety of weight reduction therapies and schemes. These range from various diet and behavioral modification programs to surgical approaches including intestinal bypass and gastric stapling procedures. The most recent approach to therapy has been the iatrogenic bezoar in the form of an inflatable gastric balloon. Although the GEGB was introduced on a wide scale, only a limited data base on such devices preceded the approval of the Garren bubble by the Food and Drug Administration in September 1985 (9–12). Since its initial marketing as the Garren–Edwards gastric bubble, this device has been used in an estimated 20,000 patients. Because of the potential placebo value of this and similar devices its use in a carefully controlled clinical trial was warranted.

This trial was designed with the primary objective of defining the effectiveness of the GEGB as an adjunctive therapy to diet and behavioral modification in the treatment of exogenous obesity. The double-blind design of this trial was an attempt to test the device in a setting where neither patients, members of the EDC, or individuals overseeing the follow-up care of study participants were aware of the presence or absence of the device. The inability of patients to uniformly predict the presence of a GEGB (Table 3) reflects the success of this design.

The results of this study suggest that the GEGB was no more effective than diet and behavioral modification alone in the treatment of exogenous obesity (Figures 2 and 3). Weight loss occurred in all three groups (SB, BS, and BB) over the 24-wk period of this study. The major weight loss occurred during the first 12 wk of this study, with a much more gradual decrease in the second 12 wk unrelated to randomization to sham or bubble. Although weight loss was more consistent in patients with GEGB, weight changes for the groups were not significantly different.

The question of the effect of compliance with diet and behavioral modification was addressed by evaluating attendance records at the EDC classes. Patients attending more than half of the 24 classes offered during this study period were considered compliant. Compliant patients did achieve a numerically larger change in BMI but this was not statistically significant (Figure 4). More important, the presence of a GEGB did not promote greater weight loss in a noncompliant patient than did diet and behavioral modification in a compliant sham patient. This reinforced the concept that the GEGB is not a substitute for this type of therapy.

A major question raised by this study is the potential effect of free therapy (i.e., no charges for the endoscopic procedures or the GEGB) on the results. Whether or not the lack of financial commitment biased the results cannot be answered based on the data collected. Our free therapy design was predi-

cated on our strong feelings that we should not charge patients for therapy that had not been proven to be effective. In a preliminary report by Hogan et al. (15), where financial commitment was required, greater effectiveness of the GEGB was not demonstrated. This variable, however, must be considered in future trials with similar devices.

With increasing use of the GEGB in the United States, a variety of complications have been reported (16–21, 30). In this study we saw the full array of these complications (Table 4). Esophageal laceration is a technical problem related to inadvertent inflation of the device in the esophagus. This appears to be related to those patients whose stomachs lie horizontally, making the angle between the esophagus and stomach more acute and preventing the free passage of the introducer tube into the stomach. Resistance to introducer tube passage or air insufflation, or pain felt by the patient during insertion and deployment, should alert the endoscopist to respond immediately. When difficulty is encountered, passage and deployment under direct vision is easily accomplished. Incomplete deployment (incomplete inflation or failure to exit the introducer) is usually a function of an improperly loaded device. Given the possibility of passage of a partially inflated bubble into the small bowel, all such devices must be immediately removed.

Irritation of the gastric mucosa leading to the development of GEs or GUs is common with the GEGB. It would appear that the relatively sharp ridges at both ends of this device are responsible for injury. We and others (22,23) have shown that there are no changes in gastric acid production and only transient alterations in gastric motility with the GEGB. The location of ulcers and erosions on the greater curvature of the stomach where the leading edge of the device was located supports the irritation theory. Gastric erosion was seen in 26% of patients and GU in 14%, occasionally requiring termination of therapy (3 of 90 patients, 3%). However, no bleeding or perforation from these ulcers was seen. Whether the concomitant use of H_2-blockers, sucralfate, or other agents would have decreased these complications is speculative, but it should be addressed in future studies with this or similar devices as there are preliminary data to support this supposition (24).

The most serious complication of this device is SBO. When the GEGB deflates and passes into the small bowel (8 of 90 patients, 9%) it usually passes spontaneously (6 of 8 patients, 75% in our study). However, in some patients it may lodge in the small bowel producing complete SBO (2 of 90 patients, 2%). It is important to note that the 2 patients in our study who developed SBO were both noncompliant and missed their scheduled removal dates at 3 mo. Original estimates of the durability of this device were quickly reassessed as deflation became an increasingly frequent event. It was this observation that led to a change in recommended dwelling time in the stomach from 4 to 3 mo. Even at 3 mo, the presence of gastric acid and dietary acids and aldehydes causes the polyurethane to become less air-tight, promoting deflation. It is clear that once in the small bowel, the GEGB must be regarded as a blunt foreign body as the same types of complications can be anticipated (25–27,29). Most important, it cannot be assumed that this device will pass harmlessly through the gastrointestinal tract, a fact made extremely clear in a recent report (19).

Complications encountered at removal of the GEGB seem related to the trauma of removal or retching that occurs as it is pulled through the esophagus, or both. The Mallory–Weiss tears encountered (10 of 90 patients, 11%) were generally minor with self-limited bleeding, none of them requiring treatment or transfusion.

Given the desire by both patients and health care professionals for a safe and effective treatment of exogenous obesity, great enthusiasm greeted the introduction of the GEGB into the field of obesity management. However, the data derived from this study confirm the preliminary reports from other centers that in a group of patients, the GEGB has no independent benefit beyond diet and behavioral modification in the management of exogenous obesity. The enthusiasm to use this device has been further diminished by the significant morbidity associated with its use (30) (Table 4). An ineffective device with this degree of morbidity is likely to have only a limited role in the treatment of obesity in its present form.

Given the criticism that can be leveled at this and other studies and the nature of obesity research, it would be inappropriate to assume that there will not ultimately be a role for this or similar devices in obesity treatment. The uniformity of weight loss in the patients in this study and others (15,28,29) with a GEGB, however, leaves open the question of whether a more resistant, longer lasting device without this degree of morbidity might not be an important adjunctive treatment for exogenous obesity. What role do such devices play at present? At a recent symposium on the treatment of exogenous obesity with iatrogenic bezoars, it was recommended that these devices should only be used in clinical trials where specific questions can be addressed (30). In concurrence with this recommendation, it is our belief that the GEGB should be limited to clinical trials where study design is the cooperative product of experts in obesity management and skilled thera-

peutic endoscopists. This recommendation should be heeded not only for the GEGB but also for the wide array of similar devices that are currently being used worldwide and that are undergoing preliminary evaluation in this country (30).

References

1. Van Itallie TB. Obesity, adverse effects on health and longevity. Am J Clin Nutr 1979;32:2733–51.
2. Bray GA. Complications of obesity. Ann Intern Med 1985;103:1052–62.
3. Health implications of obesity. National Institutes of Health Consensus Development Conference (Statement). Ann Intern Med 1985;103:147–51.
4. Kollar EG, Atkinson RM, Albin DL. The effectiveness of fasting in the treatment of superobesity. Psychosomatic 1968;10:125–35.
5. Wadden TA, Stunkard AJ, Brownell KD, Day SC. Treatment of obesity by behavior therapy and very low calorie diet. J Consult Clin Psychol 1984;52:692–4.
6. Mason EE. Evolution of gastric reduction for obesity. Contemp Surg 1982;20:17–23.
7. Adibi SA, Stanko RT. Perspectives on gastrointestinal surgery for treatment of morbid obesity: the lesson learned. Gastroenterology 1984;87:1381–91.
8. DeBakey M, Ochsner A. Bezoars and concretions: a comprehensive review of the literature with an analysis of 303 collected cases and a presentation of additional cases. Surgery 1938;4:934–63.
9. Muller JD. Intragastric prosthesis for management of obesity. World J Surg 1982;6:492–6.
10. Nieben O, Harboe H. Intragastric balloon as an artificial bezoar for the treatment of obesity. Lancet 1982;i:198–201.
11. Taylor TV, Pullan BR. Initial experience with a free floating intragastric balloon in the treatment of morbid obesity. Gut 1983;24:A979.
12. Percival ML. "The balloon diet" a noninvasive treatment for morbid obesity; preliminary report of 108 patients. Canad J Surg 1984;27:135–6.
13. Garren LR, Garren M. The Garren gastric bubble: an endoscopic aid to treatment of morbid obesity. Gastrointest Endosc 1984;30:A153.
14. Garren L, Garren M, Giordano F, et al. Further experience with the Garren–Edwards gastric bubble as an adjunctive therapy in obesity. Gastrointest Endosc 1986;32:A170–1.
15. Hogan RB, Johnson JH, Long BW, et al. The gastric bubble vs. sham endoscopy: a prospective, randomized, controlled, double-blinded comparison as an adjunct to a standard weight loss program. Gastrointest Endosc 1987;33:A172.
16. Kirby DF, Mills PR, Kellum JM, et al. Incomplete small bowel obstruction by the Garren–Edwards gastric bubble necessitating surgical intervention. Am J Gastroenterol 1987;81:251–3.
17. Fleisher A, Conti PS, McCray RS, Nay HR. Jejunal entrapment of a gastric balloon. JAMA 1987;257:930.
18. Bonefas E, Gathe JC, Sasso RD. Small-bowel obstruction due to migration of intragastric balloon. Surgical Rounds 1987;10:84–5.
19. Boyle TM, Agus SG, Bauer JJ. Small bowel obstruction secondary to obturation by a Garren gastric bubble. Am J Gastroenterol 1987;82:51–3.
20. Fedotin MS, Ginsberg BW. Partial deployment of the Garren gastric bubble: a new complication. Am J Gastroenterol 1987;82:470–1.
21. Benjamin SB. Small bowel obstruction and the Garren–Edwards bubble: lessons to be learned? Gastrointest Endosc 1987;33:A183.
22. Collen MJ, Ciarleglio CA, Stanczak VJ, et al. The effect of the Garren–Edwards gastric bubble on basal acid and pepsin output and basal serum gastrin and pepsinogen concentrations. Gastroenterology 1987;92:A1350.
23. Collen MJ, Ziessman HA, Maher KA, et al. The effect of the Garren–Edwards gastric bubble on liquid and solid gastric emptying. Gastroenterology 1987;92:A1352.
24. White S. Effect of ranitidine on the durability of Garren/Edwards gastric bubbles. Gastrointest Endosc 1987;33:A179.
25. Moriel EZ, Avalon A, Eid A, Rachmilewitz D, Krausz MM, Durst AL. An unusually high incidence of gastrointestinal obstruction by persimmon bezoars in Israeli patients after ulcer surgery. Gastroenterology 1983;84:752–4.
26. Goldstein SS, Lewis JH, Rothstein R. Intestinal obstruction due to bezoars. Am J Gastroenterol 1984;79:313–8.
27. Krausz MM, Moriel EZ, Ayalon A, Pode D, Durst AL. Surgical aspects of gastrointestinal persimmon phytobezoar treatment. Am J Surg 1986;152:526–30.
28. Meshkinpour H, Hso D, Farivar S. The effect of gastric bubble as a weight reduction device: a controlled, crossover study. Gastroenterology 1987;92:A1532.
29. Levine GM, Goldstein M, Lowe M, Scher J, Pusateri J. The gastric bubble—fad or fantastic? Gastroenterology 1987;92:A1505.
30. Schapiro M, Benjamin S, Blackburn G, et al. The intra-gastric balloon for obesity treatment—summary of a scientific workshop. Gastrointest Endosc 1987;33:323–8.

Received September 25, 1987. Accepted December 30, 1987.

Address requests for reprints to: Stanley B. Benjamin, M.D., Chief, Gastroenterology Division, Georgetown University, Room M 2118, 3800 Reservoir Road, Washington, D.C. 20007.

This study was presented to the Plenary Session of the American Society of Gastrointestinal Endoscopy, Chicago, Illinois, May 1987.

The authors thank Erna L. Benjamin for expert secretarial and editorial assistance.