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Gastroplasty for Respiratory Insufficiency of Obesity

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Three patients with the obesity hypoventilation syndrome and one patient with the sleep apnea syndrome underwent gastroplasty for weight reduction. A tracheostomy was also performed in the patient with sleep apnea. The Pa_{O_2} rose from an average of 51 ± 9 to 71 ± 5 torr and the Pa_{CO_2} fell from an average of 51 ± 12 to 41 ± 6 torr within two to ten months following bariatric surgery. The improved arterial blood gases were associated with an increased forced vital capacity in each patient. The change in maximum voluntary ventilation was variable. Sleep capneography demonstrated cure of the patient with sleep apnea permitting removal of the tracheostomy. All four patients have returned to productive lives in society. Given proper pre- and postoperative care, patients with respiratory insufficiency tolerate the operation well. Respiratory insufficiency associated with morbid obesity should be considered an indication for the gastroplasty procedure, rather than a contraindication as previously suggested.

THE PATHOPHYSIOLOGY OF respiratory insufficiency associated with excessive weight was initially studied by Burwell et al.[1] They coined the term "Pickwickian syndrome" to describe a markedly obese patient who fell asleep in a poker game while holding a hand containing three aces and two kings. This patient was somnolent, hypoxic, polycythemic and suffered from right ventricular failure, hypercapnea, periodic breathing with apneic episodes during sleep and a blunted ventilatory response to CO_2 rebreathing, all of which returned to normal after a 17 kg weight loss. It is now apparent that there are two primary forms of obesity induced respiratory insufficiency. These are the obesity hypoventilation and the sleep apnea syndromes.[2-4] Patients with the obesity hypoventilation syndrome have a restrictive pattern on pulmonary function tests with a reduced forced vital capacity (FVC) and functional residual capacity (FRC) and a marked reduction in maximal voluntary ventilation (MVV). Hypoxemia and hypercarbia are found on arterial blood gas analysis. Hypoxic pulmonary arterial hypertension is present. Most patients with the sleep apnea syndrome suffer from acute upper airway obstruction during sleep which produces hypoxemia and hypercarbia arousing the patient.[5] Both groups of patients are prone to bouts of sleepiness during normal waking hours.

Weight reduction has been shown to be effective in curing the obesity hypoventilation syndrome.[1,6] Tracheostomy will relieve the upper airway obstruction by bypassing the glottis.[7] The effect of weight loss alone on reducing upper airway obstruction in sleep apnea is variable.[8] Extensive and long lasting voluntary weight reduction by dietary means alone is usually unsuccessful.[9] The gastroplasty operation, also known as the gastric stapling or gastric partition procedure, is a new surgical approach to the treatment of morbid obesity[10,11] which does not carry the severe metabolic risks of the jejunoileal bypass[12-18] or the operative risks of the gastric bypass procedure.[19-22]

This paper describes our experience with bariatric surgery for the treatment of four patients with respiratory insufficiency secondary to obesity who had not been able to lose weight by dietary means alone. Their

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courses are contrasted to the 45 other obese patients who had gastroplasty but did not have preoperative respiratory insufficiency. One patient with severe sleep apnea suffered a respiratory arrest and died shortly after surgical evaluation.

Methods

Patients were candidates for the gastroplasty operation if they were more than 45 kg over ideal body weight and had failed to lose weight and maintain the loss through dietary management. Preoperative evaluation included arterial blood gas analysis, electrocardiogram, chest radiographs, complete blood count (CBC), chemical evaluation by sequential multiple analyzer computerized (SMAC), and urinalysis. The diagnosis of obesity hypoventilation syndrome was based on evidence of arterial hypoxemia ($Pa_{O_2} < 60$ torr) and hypercarbia ($Pa_{CO_2} > 45$ torr) on ambient room air. Patients with symptoms suggestive of the sleep apnea syndrome were evaluated by nighttime capneography. If apneic periods (cessation of airflow for >15 sec) were documented, complete polysomnographic sleep studies were performed and heart rate, electroencephalogram, respiratory effort (pneumograph) and airflow at the nose and mouth (thermistors) were monitored continuously. Sleep apnea was diagnosed according to the definition of Guilleminault.[5] Patients with hypoxemia and/or hypercarbia underwent pulmonary function testing in the sitting position which included measurement of FVC, FRC (by helium dilution), and MVV.

The first 21 patients, including one of the obesity hypoventilation patients (PW), underwent gastroplasty with a single row of staples. Because of staple line disruption in four patients, the procedure was changed to the application of a double row of staples with three staples removed from each row and a circumferential, 2-0 Prolene suture, to calibrate the stoma to 12 mm diameter. Patients were extubated in the recovery room when they were able to maintain a $Pa_{O_2} > 70$ torr and $Pa_{CO_2} < 40$ torr on a T-piece, with 40% inspired oxygen ($F_{IO_2} = 0.4$). If the patients were not able to be extubated within 12 hours, they were transferred to the surgical intensive care unit (SICU). After operation, all patients were placed in a deep semirecumbent position to maximize diaphragmatic excursion and minimize atelectasis and pulmonary shunting. Each patient was ambulated the night of the operation and encouraged to move his legs when in bed. Intravenous cephalothin was administered before operation. The wound was irrigated with either neomycin or gentamicin prior to closure and a suction drain inserted. No subcutaneous sutures were used. The skin was approximated with staples. The nasogastric tube was removed in two to four days and the diet slowly advanced from one ounce of clear liquids per hour to full liquids *ad libitum*.

An upper gastrointestinal (UGI) series was obtained prior to discharge from the hospital to determine the adequacy of the procedure and to serve as a baseline for future studies. Subsequent UGI series were obtained if clinical symptoms of staple line disruption or stomal stenosis developed. All patients were maintained on a full liquid diet for the first two months following surgery.[10] After operation the patients were seen two weeks, two months, five months, eight months, one year and then at six month intervals at which time they were weighed and blood drawn for laboratory studies. Those patients with the obesity hypoventilation or the sleep apnea syndrome underwent repeat pulmonary function testing including arterial blood gas analysis two to ten months after operation.

Results

We identified three patients with the obesity hypoventilation syndrome, all of whom had marked impairment of preoperative blood gases on multiple occasions. Two of the patients (FN, MS) received bronchodilators for moderately and mildly severe asthma, but neither patient required steroids for control of bronchospasm. The third patient (PW) smoked approximately 45 cigarettes per day, but decreased to five cigarettes per day for four weeks before operation. She resumed heavy cigarette use soon after discharge from the hospital. Two additional patients had sleep apnea identified by sleep capneography (Fig. 1) and confirmed as obstructive apnea by polysomography (Fig. 2). Preoperative arterial blood gases and pulmonary function tests in these five patients with respiratory insufficiency are shown in Table 1. Significant reductions in FRC, FVC, and MVV were noted. The average preoperative arterial blood gases in the 44 patients without respiratory insufficiency were $Pa_{O_2} = 80 \pm 12$ torr and $Pa_{CO_2} = 35 \pm 4$ torr.

The gastroplasty procedure was performed uneventfully in each of the three patients with obesity hypoventilation and in patient MM with the sleep apnea syndrome, who also underwent tracheostomy. A fifth patient (WU) had a respiratory arrest during preoperative evaluation and subsequently died. The three patients with hypoventilation of obesity all required prolonged mechanical volume ventilation in the intensive care unit ranging from 18 hours, in patient MS, to six days, in patient FN, following surgery. One patient (PW) required 10 cm H_2O positive end-expiratory pressure (PEEP) for three days to maintain her Pa_{O_2} above 60 torr with $F_{IO_2} = 0.4$. She was able to be extubated on

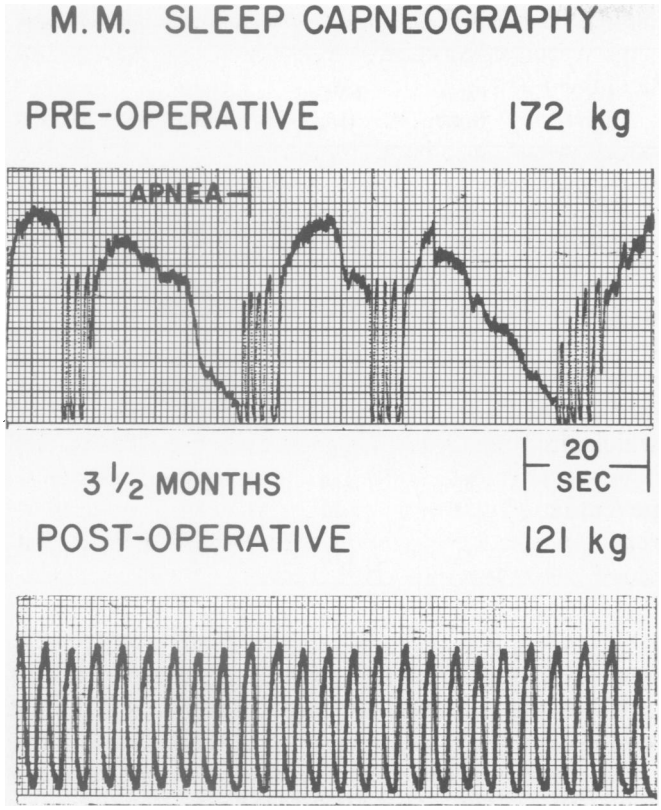

FIG. 1. Sleep capneography in Patient MM, where vertical axis is qualitative measurement of expired CO_2. Frequent apneic episodes were noted before, and absence of apnea after, gastroplasty and 51 kg weight loss.

the fourth post-operative day. Patient FN, with moderate bronchial asthma, was accidentally extubated twice during the first day after operation, developed severe hypercarbia ($PaCO_2 > 60$ torr) each time, and required emergency reintubation. She was given a continuous intravenous aminophylline infusion (1 mg/ml) to maintain her serum theophylline level between 10 and 20 μg/ml. During the first four days after operation, she required a lidocaine infusion (2 mg/min) for the suppression of premature ventricular contractions. When gastrointestinal function returned the patient was administered aminophylline tablets, 200 mg every six hours, terbutaline tablets, 2.5 mg every 8 hours, and an isoetharine mesylate inhalation aerosol, every six hours as needed. These three patients were discharged from the hospital on the sixth to thirteenth day after operation.

In contrast, all but two of the patients without hypoventilation of obesity were able to be extubated within six hours of surgery. These two patients weighed 205 and 225 kg, respectively, and were treated with a mechanical volume ventilator in an intensive care unit for 24 hours following surgery.

All four patients with preoperative respiratory insufficiency had salutary responses to gastroplasty. Within two to ten months following surgery and the loss of 17 to 51 kg body weight, marked improvements in arterial blood gases (Figs. 3 and 4) and pulmonary function tests (Table 1) were demonstrated in all four patients.

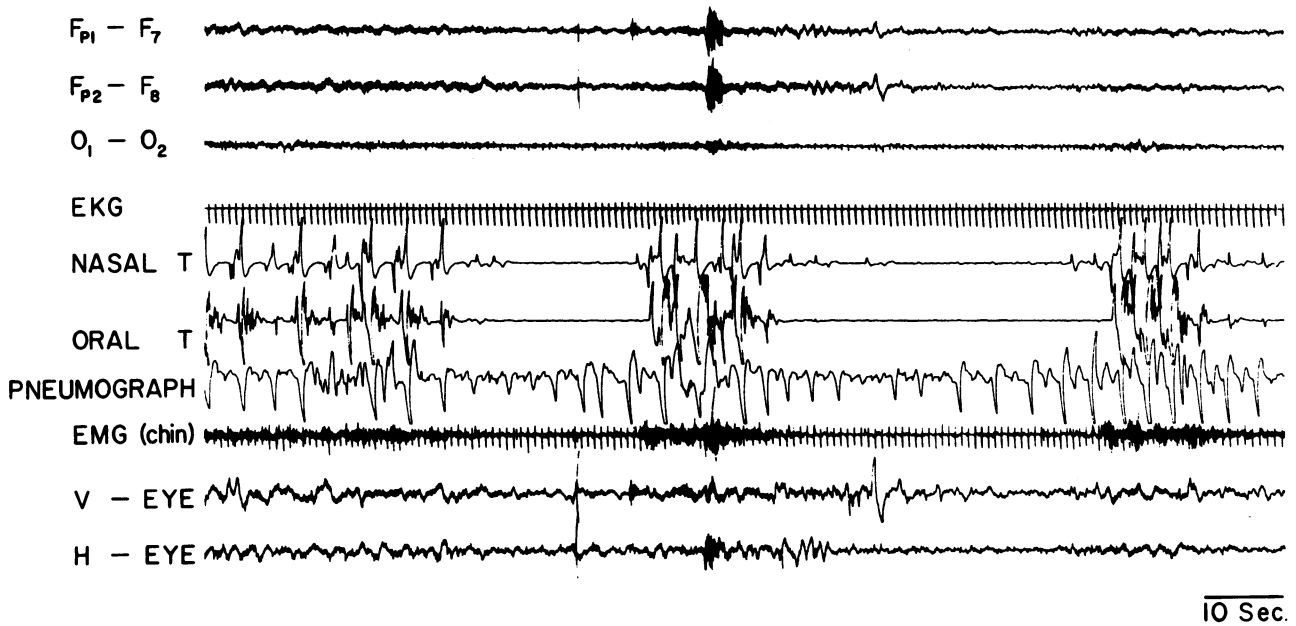

FIG. 2. Polysomnography in patient WU. Simultaneous electroencephalogram (EEG) with leads $F_{P1}-F_7$, FP_2-F_8, 0_1-0_2, electrocardiogram (EKG), nasal and oral airway temperature, pneumograph utilizing a circumferential chest belt, electromyography (EMG) of submental muscle and random eye movements (V and H) showing cessation of airflow with persistence of respiratory efforts. Patient arouses (note EEG and EMG) after 25 and 45 second apneic episodes with return of air flow.

TABLE 1. *Measurements of Weight, Arterial Blood Gases and Pulmonary Function Tests Before and After Gastroplasty Induced Weight Loss*

	Ideal Weight (kg)	Actual Weight (kg)	Pa_{O_2} (torr)	Pa_{CO_2} (torr)	FVC (L)	MVV (L/min)	FRC (L)
Obesity hypoventilation syndrome							
M.S. (56-year-old white woman)							
preoperatively	69	140	59	47	1.92	72	2.00
6 months		92	70	38	2.76	90	2.65
P.W. (44-year-old white woman)							
preoperatively	60	140	44	46	2.35	60	1.20
10 months		112	77	43	2.93	81	—
16 months		85	79	42	3.18	88	1.96
F.N. (30-year-old black woman)							
preoperatively	61	127	43	68	1.86	66	—
2 months		110	72	49	2.40	43	—
Sleep apnea syndrome							
M.M. (30-year-old white man)							
preoperatively	85	172	58	42	4.89	107	1.67
4 months		121	65	35	5.72	100	3.54
W.U. (38-year-old black man)							
preoperatively	75	156	38	56	3.60	102	1.10

Patient FN, who lost only 17 kg, had significantly improved arterial blood gases and FVC but a reduced MVV. Following weight losses of 28 and 48 kg, respectively, patients PW and MS had significant improvements in arterial blood gases, FVC, FRC and MVV (Table 1).

The response of the patient with sleep apnea was particularly gratifying. He was unable to work prior to surgery. He sold his restaurant business and his car for fear of falling asleep at the wheel. Before operation, he was frequently found in his room asleep, severely cyanotic and apneic. His snoring was legendary. He had been treated with 80–160 mg of dextroamphetamine daily in attempts to alleviate his daytime sleepiness. After operation the patient was alert throughout the day without further episodes of cyanosis. Following the loss of 51 kg in three and one half months, the patient's sleep apnea syndrome resolved completely as documented by sleep capneography with his tracheostomy plugged (Fig. 1). His tracheostomy was removed at that time and the patient has remained asymptomatic.

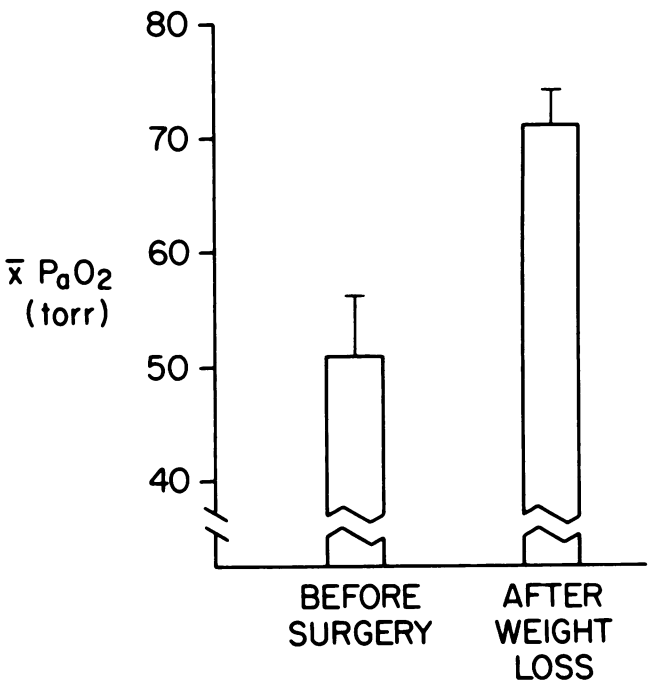

FIG. 3. Average rise in Pa_{O_2} following gastroplasty induced weight loss.

Subjectively, exercise tolerance increased in each of the four patients and each is gainfully employed.

Early complications following gastroplasty are listed in Table 2. Two of the initial 21 patients with a single row of staples developed stomal obstruction requiring early operative intervention, and one of the patients with a double row of staples developed a leak in the proximal pouch. Three of the wound infections occurred in patients without pre- or postoperative antibiotics or antibiotic irrigation of the wound. One wound infection occurred in one of the two patients with simultaneous gastroplasty and take-down of a jejunoileal bypass.

Late complications are listed in Table 3. Four of 21 patients (19%) with a single row of staples developed staple line disruption four months to one year following gastroplasty. In the past nine months, 28 patients have undergone gastroplasty with a double row of staples with the addition of a circumferential, 2-0 Prolene seromuscular suture at the stoma. Of the four patients with hypoventilation of obesity, one (PW) developed staple line disruption as well as an incisional hernia five months following her initial operation. Her pulmonary function tests prior to the second operation were

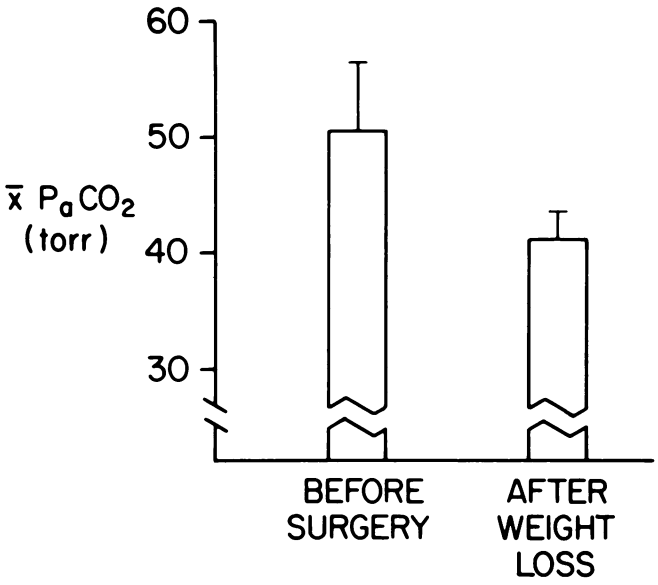

FIG. 4. Average fall in $PaCO_2$ following gastroplasty induced weight loss.

greatly improved. Despite the greater trauma of a second operative procedure, she was able to be extubated in the recovery room and did not require treatment in the intensive care unit. Three patients who complained of marked nausea and vomiting had evidence of stomal stenosis on UGI. Two of these patients were successfully dilated to 12 mm diameter using the Eder-Peustow® dilators.

In general, most patients were quite satisfied with the operation. They described not only early satiety but also a pronounced loss of appetite. Following staple line disruption in four patients, a sudden increase in appetite was noted. Their satisfaction with the initial results of the operation was apparent in their decision to undergo revision. Following correction of the staple line disruption, each of them again noted a return of early satiety as well as a marked decrease in appetite.

Neither electrolyte nor liver function test abnormalities developed in these patients following gastroplasty. The lactic dehydrogenase (LDH) was elevated in all patients before operation and may have reflected marked fatty infiltration of the liver noted on biopsy specimens. The LDH gradually decreased to normal levels after operation. The only significant metabolic abnormality noticed was hyperuricemia in 60% of the patients, which was associated with gouty arthropathy in three. The uric acid levels returned toward normal as the rate of weight loss fell.

Discussion

The increased mortality rate associated with morbid obesity has been appreciated since the writings of Hippocrates who wrote in his Aphorisms that "those naturally fat are more liable to sudden death than the thin".[4] Shakespeare must have appreciated the deleterious effects of obesity on respiration when he wrote in Hamlet, Act V: "He's fat and scant of breath." Since the detailed case report of Burwell et al.,[1] where the term "Pickwickian Syndrome" was coined, a number of studies of the effects of obesity on ventilation have been performed.[2–4] The somnolence was initially thought to be secondary to hypoxemia. Although this appears to be the primary pathophysiology in most of these patients, others suffer from the sleep apnea syndrome. These patients have glottic upper airway obstruction during sleep and become apneic preventing an adequate rest so that they suffer from severe drowsiness during normal waking hours. This syndrome can be documented by sleep capneography and polysomnography (Figs. 1 and 2).

The obesity hypoventilation syndrome is thought to be a result of decreased chest wall compliance and an elevated diaphragm, both of which decrease FRC so that tidal ventilation reaches alveolar closing volumes at end expiration producing pulmonary arteriovenous shunting and hypoxemia.[2] In addition, the hypoxemia produces polycythemia and an increased pulmonary blood volume which may further decrease pulmonary compliance.[2] With the presence of additional pulmonary disease the obese hypoxic patient develops hypercarbia with a reduced responsiveness to carbon dioxide.[1,6] All three of our patients with the obesity hypoventilation syndrome had superimposed pulmonary disease. Two of them suffered from bronchial asthma and one patient had cigarette induced chronic obstructive pulmonary disease. Obesity was much greater in a number of our patients who did not have respiratory insufficiency.

There have been a number of publications regarding the changes in arterial blood gases and pulmonary func-

TABLE 2. *Early Complications Following Gastroplasty in 49 Patients*

Stomal obstruction	2
Perforation	1
Dehiscence	1
Wound infection	5
Incidental splenectomy	4

TABLE 3. *Late Complications Following Gastroplasty in 49 Patients*

Staple disruption*	4
Stomal stenosis†	3
Incisional hernia	4
Gout	3
Lost to follow-up	3

* All in patients (21) with single row of staples.
† Two patients endoscopically dilated to 12 mm diameter.

tion tests following weight loss after the jejunoileal bypass procedure in patients without the obesity hypoventilation syndrome. In most of these patients, FRC[23,24] and MVV increased following surgery.[25] Some patients had a small but significant increase in Pa_{O_2},[23,26–28] whereas others showed negligible changes.[24,25,29] Some authors have stated that obesity induced respiratory insufficiency may be a contraindication to surgery for morbid obesity.[24] Because the gastroplasty operation could be performed expeditiously with few complications, it seemed appropriate to offer this procedure to patients who were incapacitated by respiratory insufficiency. Four of these patients underwent the operation uneventfully. Only one patient had difficulty in being weaned from the ventilator. Objective data confirmed both marked improvement in Pa_{O_2} (Fig. 3) and reduction in CO_2 retention (Fig. 4) following weight loss. There was a significant improvement in FVC in each of the patients and in FRC in the three patients in whom it was measured before and after surgery (Table 1).

Patients with the sleep apnea syndrome may require tracheostomy.[7] This bypasses the glottic upper airway obstruction that occurs with sleep which is responsible for the apneic episodes and hypoxemia. In a study by Guilleminault et al.,[8] weight loss improved the symptoms but did not completely resolve the apneic episodes. In patient MM, however, weight reduction from 172 to 121 kg over three and one half months, resulted in complete cessation of his apneic periods (Fig. 1). His tracheostomy was removed at that time and he remains asymptomatic.

Although the gastroplasty operation is technically easy to perform, there are a number of complications that can occur which might be fatal in a patient with respiratory insufficiency. These include perforation of the gastric pouch, acute stomal obstruction, pulmonary embolism, atelectasis and pneumonia.[10,11,20,34] Every effort should be made to improve pulmonary function before operation. Cigarette smoking should be stopped at least one month before surgery. Excessive fluid should be removed and cardiac function improved with diuretics and digitalis when indicated. Preoperative instruction in pulmonary toilet and "incentive spirometry" should be given. These patients must be carefully monitored in an intensive care unit. It has been shown that pulmonary function is markedly enhanced by placing the patient in a semirecumbent position.[30,31] This permits increased diaphragmatic excursion and maximal FRC. PEEP should be used with caution since it can paradoxically lower Pa_{O_2} by reducing blood flow to well ventilated lung units.[4] It was of value, however, in one of our patients. It is important that the patients move their legs and feet frequently to reduce venous stasis and the dangers of venous thrombosis and pulmonary embolism. Patients with severe hypoxemic pulmonary hypertension should be considered for a prophylactic inferior vena caval filter inserted preoperatively since a small pulmonary embolus could be fatal in such a patient.[32] All patients, including those requiring mechanical ventilation, should be ambulated the night of the operation.

It is clear from our study as well as several others[22,33,34] that a single row of staples is prone to disruption, despite institution of a liquid diet for the first eight weeks following surgery. In addition, stomal dilatation can occur if it is not adequately reinforced. For this reason, we have chosen to insert two rows of staples as well as a running, circumferential suture of 2-0 prolene at the stoma. The high incidence of stomal stenosis or obstruction (five of 49 patients) may have resulted from mucosal damage produced when the TA-90 stapling device was approximated. The placement of the stoma on the greater curvature with the use of the Gomez C-clamp may reduce the incidence of this complication.[11]

The only metabolic complication seen after gastroplasty in our patients was the development of hyperuricemia associated with the early phase of rapid weight reduction. Three patients developed acute gouty arthropathy. There were no electrolyte, liver or renal function abnormalities. Although this operation is far safer than the jejunoileal intestinal bypass, a number of deaths have been reported secondary to either a gastric leak or pulmonary embolism.[10,11,20,33] The operation should not be undertaken lightly.

Although several of our patients have complained of excessive nausea and vomiting, the majority have achieved comfortable early satiety as well as a marked decrease in appetite. This relative anorexia disappeared acutely at the time of staple line disruption in four patients, but returned upon surgical revision. Further investigation into the physiology of this response might provide clues to the medical treatment of morbid obesity.

The cure of the hypoventilation syndrome in three patients and the sleep apnea syndrome in one patient suggests that these should be indications for the gastroplasty procedure in morbid obesity rather than contraindications as previously suggested. With careful pre- and postoperative care, these patients tolerate the operation well and show improvement in objective measurements of ventilatory function. Consequently, our patients not only improved clinically but were able to return to productive lives in society.

Acknowledgments

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Discussion

Dr. Richard J. Field, Jr. (Centreville, Mississippi): I heartily agree with Dr. Greenfield. In our experience, it's paradoxical that the very patients that are turned down in many institutions today because they have this severe obesity hypoventilation syndrome are the very ones who, indeed, need to have it more than any others. And I would like to present to Dr. Greenfield two problems and get his opinion upon them.

At the risk of exposing my own ineptness, we find, occasionally, in this type work, that we run into a Gargantua-like individual and we're not able to safely reach the esophagogastric junction. (slide) We find that in some of these people with huge livers we are unable to actually reach the esophagogastric junction, even with all of the new type obesity retractors. This fellow presents such an interesting problem. He is 36 years old and a Grade IV obesity hypoventilation syndrome case. Just to make it interesting, he has an idiopathic lymphedema in his left leg. He's a hypertensive, in chronic congestive heart failure, hypercarbic and his plasma oxygen was very low.

He had been turned down for obesity surgery by two institutions, and you can see why; but, actually, their reasons were the ones that Dr. Greenfield has mentioned so well. This fellow had no exercise tolerance. He slept nearly all the time and was unable to work.

So he came to us, and we felt almost surely we would not be able to reach his esophagogastric junction; and, sure enough, we were not, although we tried rather valiantly. We had prepared him for this possibility and had advised him of our plan to do an intestinal bypass with the idea of later doing a gastroplasty when he had lost much weight.

(slide) This is a side view. I didn't tell you that he weighed 639 pounds. We performed an intestinal bypass. (slide) In two years' time he had lost 409 pounds; however, he was totally undisciplined. The patient would not take any of his medicine, and he became hypokalemic and hypocalcemic, although his heart size had returned to normal and his pulmonary function studies were normal. At this point he was also hypoproteinemic and very weak. We chose then to do a gastroplasty, which brings up another problem.

(slide) We did a 50 cc proximal pouch gastroplasty, which is ideal and has been described by most people doing this work. However, I think it is a mistake. It has been our observation that the vast