

Laparoscopic Gastric Banding Prevents Type 2 Diabetes and Arterial Hypertension and Induces Their Remission in Morbid Obesity

A 4-year case-controlled study

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OBJECTIVE — Lifestyle modifications and pharmacological interventions can prevent type 2 diabetes in obese subjects with impaired glucose tolerance. The aim of this study was to compare laparoscopic adjustable gastric banding (LAGB) and conventional diet (No-LAGB) in the prevention (primary intervention study; 56 vs. 29 patients) and remission (secondary intervention study; 17 vs. 20 patients) of type 2 diabetes and hypertension in grade 3 obesity in a 4-year study.

RESEARCH DESIGN AND METHODS — The subjects ($n = 122$; age 48.5 ± 1.05 years; BMI 45.7 ± 0.67 kg/m²) underwent a diagnostic workup, including psychological and psychiatric assessments, in preparation for the LAGB procedure. Of the 122 subjects, 73 had the surgery (LAGB group). The control group (No-LAGB group) consisted of the 49 subjects who refused the surgery but agreed to be followed up; 6 of these subjects dropped out by the 2nd year of the study, so that the final number of patients was 73 and 43 in the LAGB and No-LAGB groups, respectively. All patients had a yearly visit and oral glucose tolerance test.

RESULTS — From baseline to the end of the 4-year follow-up, BMI decreased from 45.9 ± 0.89 at baseline to 37.7 ± 0.71 kg/m² in the LAGB group and remained steady in the No-LAGB group (from 45.2 ± 1.04 to 46.5 ± 1.37 kg/m²), with no significant differences between the primary and secondary intervention groups. In the primary intervention study, five of the No-LAGB subjects (17.2%) and none of the LAGB subjects (0.0%; $P = 0.0001$) progressed to type 2 diabetes; in the secondary intervention study, type 2 diabetes remitted in one No-LAGB patient (4.0%) and seven LAGB patients (45.0%; $P = 0.0052$). Hypertension occurred in 11 No-LAGB patients (25.6%) and 1 LAGB patient (1.4%; $P = 0.0001$) and remitted in 1 No-LAGB (2.3%) and 15 LAGB patients (20.5%; $P = 0.0001$). A study of body mass composition revealed a significant reduction of fat mass and a transitory, but not significant, decrease of fat-free mass in LAGB patients.

CONCLUSIONS — In morbid obesity, sustained and long-lasting weight loss obtained through LAGB prevents the occurrence of type 2 diabetes and hypertension and decreases the prevalence of these disorders.

Diabetes Care 28:2703–2709, 2005

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Received for publication 11 April 2005 and accepted in revised form 10 August 2005.

Abbreviations: BPD, biliary pancreatic diversion; GBP, gastric bypass; IGT, impaired glucose tolerance; LAGB, laparoscopic adjustable gastric banding; NGT, normal glucose tolerance; OGTT, oral glucose tolerance test; SOS, Swedish Obese Subjects.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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Obesity is a major risk factor for certain diseases, particularly cardiovascular disease. The risk is proportional to BMI and duration of obesity and increases with visceral obesity (1–6). Obesity, especially when associated with impaired glucose tolerance (IGT), is a leading cause of type 2 diabetes (7). Several large studies have demonstrated that it is possible to prevent the progression from IGT to type 2 diabetes by dietary intervention, lifestyle modifications (including physical activity), and drugs (8–13). In some cases, these therapeutic approaches reduce cardiovascular morbidity and mortality in type 2 diabetes (14,15). Obesity and type 2 diabetes are often complicated by arterial hypertension, a link that is allegedly mediated by increased sympathetic tone (16); left ventricular hypertrophy is found in long-standing obesity (4) and/or arterial hypertension (17) and is the supposed mechanism for congestive heart failure in obesity (18).

Bariatric surgery is performed in morbid (grade 3) obesity, when BMI is >40 kg/m² in the absence of comorbidities or >35 kg/m² in the presence of type 2 diabetes or hypertension (19). In several studies, laparoscopic adjustable gastric banding (LAGB), gastric bypass (GBP), and biliary pancreatic diversion (BPD) decrease the prevalence of type 2 diabetes and hypertension, showing progressively greater efficacy (20–27). The different efficacy with type 2 diabetes seen across the studies may be attributable to the different levels of weight loss, which was greater for BPD than for LAGB or GBP (24) and similar or slightly superior for GBP than for LAGB (24,25), and to the different criteria for defining reduced prevalence of type 2 diabetes, whether it was fasting blood glucose and HbA_{1c} (A1C) levels (20,22–24) or oral glucose tolerance tests (OGTTs) (21). In addition, two studies, mainly based on GBP and vertical banded gastroplasty, have shown

Table 1—Clinical and metabolic characteristics of patients at entry into the study

	LAGB	No-LAGB	P
Primary intervention study			
n (men/women)	56 (8/48)	29 (5/24)	0.97
Age (years)	46.1 ± 1.48 (43.1–49.1)	44.4 ± 2.12 (40.0–48.7)	0.50
BMI (kg/m ²)	45.2 ± 1.07 (42.9–47.3)	45.2 ± 1.61 (41.9–48.5)	0.97
Body weight (kg)	119.5 ± 3.12 (113.2–125.7)	122.0 ± 5.31 (110.9–133.1)	0.67
Glucose tolerance (NGT/IGT)	36/20	19/10	0.90
Arterial hypertension (yes/no)	19/37	12/17	0.66
Fasting blood glucose (mmol/l)	5.4 ± 0.12 (5.2–5.7)	5.4 ± 0.19 (5.0–5.8)	0.96
Fasting insulin (μU/ml)	18.1 ± 1.74 (14.4–21.6)	16.0 ± 1.36 (13.1–18.9)	0.41
A1C (%)	6.1 ± 0.11 (5.9–6.3)	6.2 ± 0.15 (5.9–6.4)	0.47
Systolic blood pressure (mmHg)	132.3 ± 1.93 (128.4–136.2)	136.9 ± 2.88 (131.2–142.1)	0.19
Diastolic blood pressure (mmHg)	83.5 ± 0.98 (81.5–85.5)	84.5 ± 1.19 (82.0–86.5)	0.55
Secondary intervention study			
n (men/women)	17 (4/13)	20 (14/6)	0.019
Age (years)	53.3 ± 1.88 (47.9–56.5)	58.2 ± 2.24 (53.5–62.9)	0.08
BMI (kg/m ²)	48.3 ± 1.51 (45.1–52.6)	45.2 ± 1.12 (42.9–47.6)	0.10
Body weight (kg)	130.1 ± 5.18 (116.7–143.6)	119.7 ± 3.95 (111.3–128.0)	0.08
Type 2 diabetes	17	20	—
Arterial hypertension (yes/no)	12/5	15/5	0.97
Fasting blood glucose (mmol/l)	9.3 ± 0.43 (7.9–10.5)	9.1 ± 0.89 (7.2–11.0)	0.83
Fasting insulin (μU/ml)	19.0 ± 3.36 (15.5–20.39)	19.3 ± 0.85 (15.6–22.9)	0.88
A1C (%)	9.4 ± 0.62 (8.0–10.6)	8.6 ± 0.42 (7.7–9.4)	0.23
Systolic blood pressure (mmHg)	143.3 ± 4.36 (129.5–153.1)	149.1 ± 3.71 (141.1–156.9)	0.19
Diastolic blood pressure (mmHg)	87.5 ± 3.22 (80.3–96.9)	86.9 ± 1.93 (82.8–90.8)	0.55

Data are means ± SE (95% CI) or absolute frequencies.

that bariatric surgery can prevent type 2 diabetes (26,27). The aim of this study was to evaluate the effect of weight loss obtained through LAGB in the prevention (primary intervention) and remission (secondary intervention) of type 2 diabetes and arterial hypertension in morbid obesity.

RESEARCH DESIGN AND METHODS

Since June 1996, LAGB (LAP-BAND; Inamed, Santa Barbara, CA) has been performed at Istituto Clinico Sant Ambrogio, Ospedale San Paolo, and Ospedale San Raffaele, in Milan, Italy, in patients with morbid obesity (i.e., grade 3 obesity as determined by World Health Organization criteria) (19,21,28).

In this study, we considered obese patients who were recruited in 1999 and followed for 4 years. According to the common protocol approved by the local ethics committees, patients were eligible for LAGB if they met the following criteria: age 18–66 years inclusive and BMI >40.0 kg/m² alone or >35.0 kg/m² in the presence of comorbidities (19,21,28). The clinical protocol, including a psychological and psychiatric evaluation, has been previously described in detail (21,29,30). Two 75-g OGTTs were per-

formed in all patients, with blood glucose levels being determined at 0 and 120 min; glucose tolerance was labeled as normal (NGT), impaired (IGT), or diabetic (31). Blood pressure was measured twice on each occasion using a sphygmomanometer with an adequate cuff and after an adequate resting period. Patients were considered to be hypertensive if their systolic/diastolic blood pressure was >140/90 mmHg (World Health Organization criteria) or when they were taking stable antihypertensive medication.

After their diagnostic workup was completed, 73 eligible patients (12 men and 61 women, age 47.5 ± 1.21 years, BMI 45.9 ± 0.88 kg/m²) underwent LAGB; 49 eligible patients (19 men and 30 women, age 50.0 ± 1.83 years, BMI 45.2 ± 1.03 kg/m²) refused LAGB for personal reasons but continued in the follow-up study and were considered as the control group (No-LAGB). All surgical procedures were performed by two staff senior surgeons. The clinical and dietary follow-up protocol has been described elsewhere (21).

Obese patients were divided according to the absence (primary intervention study) or presence (secondary intervention study) of type 2 diabetes (Table 1).

BMI, A1C, and glucose tolerance (determined by OGTT) (31) were evaluated at yearly intervals. Arterial hypertension was scored as present or absent; during the 4-year follow-up study, obese hypertensive subjects were instructed to continue their antihypertensive treatment unless symptomatic hypotension (withdrawal of treatment) or worsening of arterial hypertension (increase of treatment) occurred.

Blood glucose, insulin, and A1C levels were measured as previously described (21,32). Whole-body resistance and reactance (capacitance) were measured using a tetrapolar bioelectrical impedance analyzer (SoftTissue Analyzer; Akern Bioresearch, Firenze, Italy); data were analyzed with the Bodygram software for Windows 95–98-NT (33).

Data are given as means ± SE or frequencies. Patients were considered as long as they remained in the study. Two-sided *t* tests, repeated-measures ANOVA, reporting *F* for the interaction between time and groups, and χ^2 tests were used to analyze the differences between the groups at baseline and during follow-up. Survival curves were calculated to estimate the cumulative incidence and the remission of type 2 diabetes and arterial

Table 2—Clinical characteristics of the primary and secondary intervention subjects over the course of the study and follow-up period

	n	Basal	1 year	2 years	3 years	4 years	Significance*
Body weight (kg)							
Primary							
LAGB	56	119.5 ± 3.12	97.6 ± 2.11	94.8 ± 2.14	94.1 ± 2.32	96.4 ± 2.26	—
No-LAGB	29	116.8 ± 4.79	114.2 ± 4.67	116.2 ± 5.32	117.5 ± 5.83	119.1 ± 6.49	F = 20.33, P = 0.0001
Secondary							
LAGB	17	133.6 ± 7.18	117.7 ± 7.12	114.6 ± 7.01	115.2 ± 6.58	116.1 ± 6.59	—
No-LAGB	20	119.7 ± 3.95	119.4 ± 3.73	120.3 ± 3.69	120.4 ± 3.78	121.8 ± 3.91	F = 12.75, P = 0.0001
BMI (kg/m ²)							
Primary							
LAGB	56	45.2 ± 1.07	37.0 ± 0.75	35.9 ± 0.69	35.6 ± 0.75	36.5 ± 0.73	—
No-LAGB	29	45.2 ± 1.61	44.2 ± 1.54	45.7 ± 1.81	45.9 ± 2.04	46.5 ± 2.34	F = 20.00, P = 0.0001
Secondary							
LAGB	17	48.3 ± 1.51	41.9 ± 1.56	41.1 ± 1.50	41.2 ± 1.55	42.1 ± 1.67	—
No-LAGB	20	45.2 ± 1.12	45.3 ± 1.22	45.9 ± 1.01	46.0 ± 1.06	46.4 ± 1.17	F = 3.46, P = 0.0005
A1C (%)							
Primary							
LAGB	56	6.1 ± 0.11	5.9 ± 0.06	5.4 ± 0.09	5.3 ± 0.1	5.3 ± 0.11	—
No-LAGB	29	6.2 ± 0.16	6.0 ± 0.15	6.1 ± 0.25	5.9 ± 0.28	5.9 ± 0.28	F = 5.13, P = 0.0001
Secondary							
LAGB	17	9.4 ± 0.62	7.3 ± 0.49	7.0 ± 0.61	8.0 ± 0.74	8.0 ± 0.92	—
No-LAGB	20	8.6 ± 0.42	8.5 ± 0.37	8.5 ± 0.38	8.5 ± 0.39	8.6 ± 0.42	F = 12.09, P = 0.0001
Systolic blood pressure (mmHg)							
Primary							
LAGB	56	132.3 ± 1.93	131.5 ± 2.05	131.2 ± 2.09	129.1 ± 1.95	128.3 ± 2.36	—
No-LAGB	29	136.9 ± 2.88	137.1 ± 2.89	139.3 ± 2.97	140.7 ± 3.01	149.3 ± 4.42	F = 9.31, P = 0.0001
Secondary							
LAGB	17	143.3 ± 4.36	137.2 ± 4.28	133.2 ± 2.98	129.1 ± 4.09	128.2 ± 2.96	—
No-LAGB	20	149.1 ± 3.71	147.8 ± 3.31	148.3 ± 3.28	149.4 ± 2.91	150.6 ± 2.74	F = 3.88, P = 0.0073
Diastolic blood pressure (mmHg)							
Primary							
LAGB	56	83.5 ± 0.98	81.9 ± 1.16	81.6 ± 1.23	80.9 ± 1.19	82.0 ± 1.27	—
No-LAGB	29	84.5 ± 1.19	84.8 ± 4.22	86.9 ± 1.36	87.4 ± 1.32	92.4 ± 2.43	F = 6.73, P = 0.0001
Secondary							
LAGB	17	87.5 ± 3.22	87.0 ± 3.28	85.9 ± 3.62	83.6 ± 3.31	80.4 ± 3.05	—
No-LAGB	20	86.9 ± 1.93	86.9 ± 1.81	87.5 ± 1.95	88.9 ± 1.65	90.0 ± 0.99	F = 3.67, P = 0.0077

Data are means ± SE. Number of subjects in the No-LAGB group was 49 at baseline; 6 subjects were lost to follow-up by the 2nd year of the study. *ANOVA for repeated measures. F, interaction between time and group.

hypertension. The difference between the groups in the incidence and remission of diabetes and hypertension was tested by means of the two-sided log-rank test. All analyses of end points were based on the intention-to-treat principle. Stata's survival analysis (Stata Statistical Software, Release 8.0; Stata, College Station, TX) was used to derive the basic estimates, such as the Kaplan-Meier product-limit survivor function, testing equality of survivor functions, and incidence rate comparisons. The proportional hazard assumption was tested by the Schoenfeld option. Only six patients (4.9%) had censored times. To estimate the incidence as well as the remission of diabetes and hy-

pertension, subjects were scored at the yearly follow-up with 0 if the condition was not present or 1 if it was. For each subgroup, the proportion of subjects who developed (or recovered from) diabetes and hypertension was calculated. P values <0.05 were considered statistically significant.

RESULTS

Surgical outcomes

Stoma regulation was required in 43 of the 73 patients (1–10 regulations/patient; 1.8 ± 0.24 in the entire group). Reintervention was required in eight patients (laparotomic in six patients) due to slip-

page and pouch dilation. Removal of the LAGB was performed in four patients: one at 1 year, one at 3 years, and two at 5 years, i.e., after completion of the study.

Clinical and metabolic outcomes

The clinical and metabolic variables of patients undergoing surgery (LAGB group) and refusing surgery (No-LAGB group) did not differ at the subjects' entry into the study (Table 1), except for a slight difference in the sex ratio in the secondary intervention study. By the 2nd year of the study, six No-LAGB patients were lost to follow-up.

In the primary intervention study, body weight, A1C, and systolic and dia-

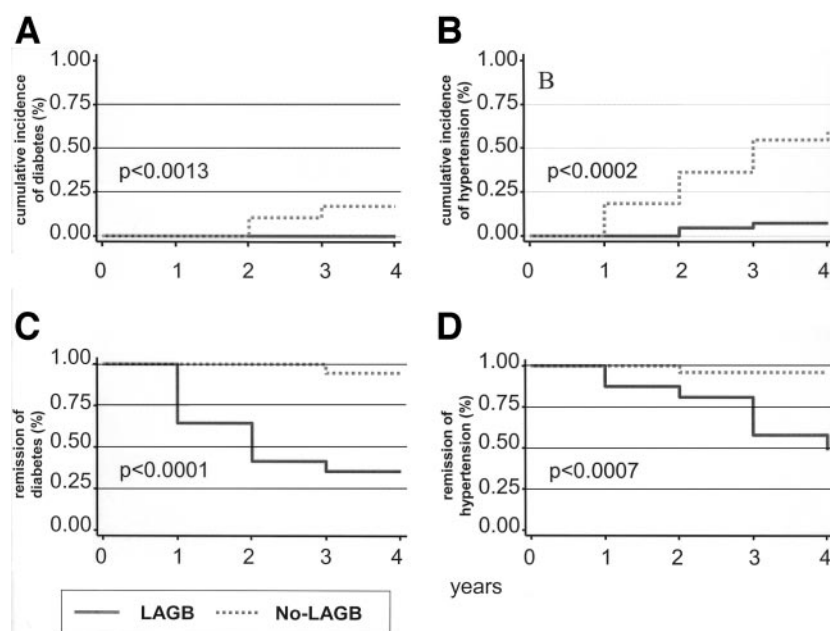


Figure 1—Cumulative incidence of type 2 diabetes (A) and arterial hypertension (B) by study group in obese patients who did not have diabetes or hypertension at entry into the study. Remission of diabetes (C) and hypertension (D) by study group in obese patients with diabetes or hypertension at entry into the study. A: Log-rank $\chi^2 = 10.29$, $P < 0.0013$; B: log-rank $\chi^2 = 13.87$, $P < 0.0002$; C: log-rank $\chi^2 = 16.05$, $P = 0.0001$; D: log-rank $\chi^2 = 11.55$, $P < 0.0007$.

stolic blood pressure significantly decreased in the LAGB but not in the No-LAGB group (Table 2). Figure 1 shows that type 2 diabetes developed during the 4-year follow-up period in none of the LAGB patients (0.0%) but in five of the No-LAGB patients, all of whom had IGT at baseline (17.2%). Figure 1 also shows that arterial hypertension developed in one patient in the LAGB group (2.5%) and in eight patients in the No-LAGB group (27.6%). Kaplan-Meier survival estimates showed that type 2 diabetes and hypertension appeared at a rate of 4.0 and 16.0 per 100 person-years, respectively, in the No-LAGB patients and 0.0 and 1.0 per 100 person-years, respectively, in the LAGB patients.

In the secondary intervention study, body weight, A1C, and systolic and diastolic blood pressure significantly decreased in the LAGB group but not in the No-LAGB group (Table 2). Remission of type 2 diabetes occurred in 45% of LAGB patients and 4% of No-LAGB patients; remission of arterial hypertension occurred in 51% of LAGB patients and 4% of No-LAGB patients (Fig. 1). Kaplan-Meier survival estimates showed that type 2 diabetes and arterial hypertension decreased at the rate of 1.2 and 1.8 per 100 person-years, respectively, in No-LAGB patients and 26.7 and 20.2 per 100 per-

son-years, respectively, in LAGB patients. Figure 2 shows changes in BMI, A1C, systolic and diastolic blood pressure, fat-free mass, and fat mass in the whole cohort of LAGB and No-LAGB patients during the 4-year study. BMI, A1C, systolic and diastolic blood pressure, and fat mass stably and significantly decreased in the LAGB as compared with the No-LAGB group; fat-free mass showed a temporary, but not significant, decrease in the LAGB group and was unchanged in the No-LAGB group.

In the LAGB group, from baseline to the 4th year of the study, glucose tolerance deteriorated (from NGT to IGT) in 1 patient (1.4%), improved (from IGT to NGT and from type 2 diabetes to IGT or NGT) in 20 patients (27.4%), and remained unchanged in 52 patients (71.2%). In contrast, in the No-LAGB group available for follow-up ($n = 43$), glucose tolerance deteriorated (from NGT to IGT or from IGT to type 2 diabetes) in 8 patients (18.6%), improved (from IGT to NGT, and from type 2 diabetes to IGT or NGT) in 4 patients (9.3%), and remained unchanged in 31 patients (72.1%). The difference between the groups was statistically significant ($\chi^2 = 14.65$, $P = 0.0007$).

Arterial hypertension appeared de novo in 1 LAGB patient (1.4%; plus tran-

siently in another LAGB patient) and in 11 No-LAGB patients (25.6%); disappeared in 15 LAGB patients (20.5%) and 1 No-LAGB patient (2.3%); and remained unchanged in 57 LAGB (78.1%) and 31 No-LAGB (72.1%; $\chi^2 = 21.97$, $P = 0.0001$) patients.

CONCLUSIONS— In this study, the weight loss obtained through LAGB prevented type 2 diabetes in morbidly obese patients for at least 4 years. LAGB also prevented the incidence of arterial hypertension and actually decreased the prevalence of hypertension already present. In patients with type 2 diabetes at entry into the study, the remission of type 2 diabetes and arterial hypertension was more frequent in the LAGB than in the No-LAGB group. In both the primary and the secondary intervention studies, the pattern of body weight, A1C, and arterial blood pressure was significantly different between the LAGB and No-LAGB groups. Therefore, these data suggest that LAGB is effective in preventing and promoting the remission of established type 2 diabetes and arterial hypertension.

An important concern in massive weight loss is that, together with fat mass, fat-free mass might also be reduced (33). We evaluated body composition through bioelectrical impedance analysis, a method that has a reliability comparable with that of other methods, such as anthropometric measurements and deuterium oxide dilution (34). As previously shown for biliary diversion (35), we observed an impressive reduction of fat mass and a transitory and not significant reduction of fat-free mass.

In some studies, BPD and, to a lesser extent, GBP have been shown to be more effective than LAGB in inducing weight loss and promoting the remission of type 2 diabetes and hypertension (20–25). However, these studies used different criteria to establish decreased prevalence of type 2 diabetes, measuring efficacy with either fasting blood glucose and A1C levels (20,22–24) or OGTTs (21). In this study, we showed that LAGB is effective in preventing type 2 diabetes, as has already been described for GBP and vertical-banded gastroplasty (26,27). In the Swedish Obese Subjects (SOS) study (27), the presence or absence of type 2 diabetes was verified only through patient self-report, which, without a direct assessment with OGTT, does not allow improvements in glucose tolerance to be ascertained. The difference between

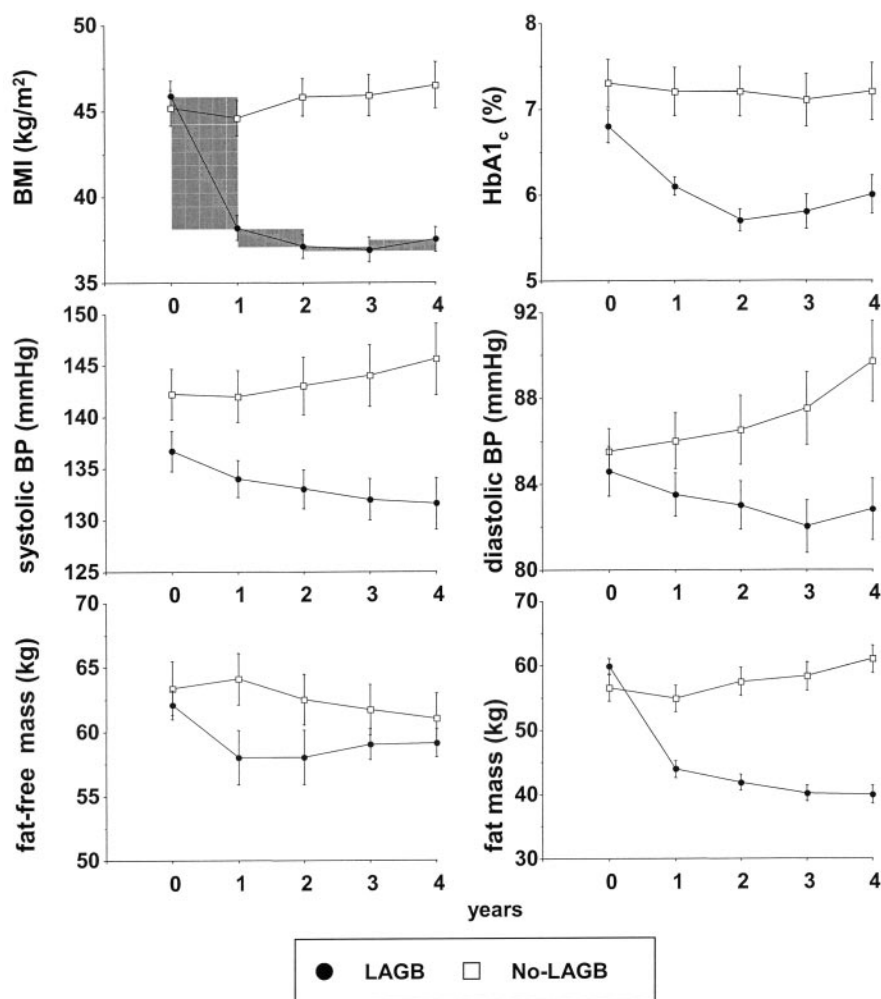


Figure 2—BMI, A1C, systolic and diastolic blood pressure, fat-free mass, and fat mass in the whole cohort of morbidly obese patients undergoing ($n = 73$; ●) and not undergoing ($n = 43$; □) LAGB. Data are means \pm SE. BMI ($F = 34.79$, $P < 0.0001$), A1C ($F = 10.11$, $P < 0.0001$), systolic blood pressure ($F = 9.89$, $P < 0.0001$), diastolic blood pressure ($F = 7.78$, $P < 0.0001$), and fat mass ($F = 28.75$, $P < 0.0001$) changed in a significant way in the two groups (ANOVA for repeated measures). Free-fat mass did not change in a significant manner.

LAGB and GBP is that the former is a minimally invasive surgical procedure, is reversible, and has a negligible surgery-related morbidity and mortality (20–27). LAGB requires some stoma regulations and occasional reinterventions, and a few patients even require or ask for the removal of the banding (21,22,25).

With regard to the prevention or improvement of arterial hypertension, we noted remission of arterial hypertension in a fair proportion of patients, similar to that reported in other studies (20–25). In contrast, the SOS study showed that gastric surgery prevented arterial hypertension at 2 but not at 8 years (27). This difference is likely due to the crucial role of body weight in the disease; in the SOS study, a significant regain of body weight was observed in surgery patients, whereas

in the present study, weight loss remained stable at $\sim 20\%$ in the primary intervention study.

Similar to other studies involving surgery, this study was not randomized; however, all our patients underwent the same diagnostic workup and made the decision to undergo or refuse LAGB. Randomized, but not blinded, surgical studies could be both feasible and ethical and would further support these results. Before this study, evidence that bariatric surgery is beneficial for type 2 diabetes and hypertension was available only for gastric bypass, vertical-banded gastroplasty, and biliary diversion (20,23–25).

Pharmacological and lifestyle modifications can prevent type 2 diabetes (8–12). The limit to this approach is that these approaches work as long as they are

applied, and it can be questioned whether they prevent or simply postpone type 2 diabetes (11,13). Another difference between pharmacological and lifestyle modifications compared with surgery is the efficacy of type 2 diabetes prevention. First, it seems that the annual incidence rate of new cases of type 2 diabetes is higher in subjects with BMI 31–34 kg/m² than in those with grade 3 obesity; this difference may also be due to the different ages of patients in the studies (8–12,26,27). Second, in some studies (8–12), risk reduction was 31–58% (average 45%), meaning that $\sim 55\%$ treated patients became diabetic. In other studies (26,27) and this study, risk reduction was 80–100% (average 90%), meaning that $<20\%$ of treated patients became diabetic. From this viewpoint, surgery is preferable in morbid obesity, as it is effective for at least 10 years (LAGB) or indefinitely (gastric bypass). The duration of type 2 diabetes is a risk factor for complications as is the duration of obesity (e.g., left ventricular hypertrophy) (4,36). It remains to be shown if these complications are prevented by weight loss obtained through LAGB, although evidence exists to this effect with regard to the reduction of total and cardiovascular mortality for GBP (37,38).

This study had some limitations. First, compared with intervention studies based on dietary restrictions, drugs, and/or lifestyle modifications, our study was relatively small; however, it should be recalled that morbid obesity represents $\sim 5\%$ of all cases of obesity (39). Second, even though the rate of dropouts was low in this study, obesity studies are usually affected by high rates of dropouts, whether they are diet (40) or surgery (23) studies. Third, this was an observational study, not a randomized clinical trial, but we have no reason to suspect that the large differences between the groups were the result of confounding factors.

In conclusion, this study showed that in grade 3 obesity, weight loss obtained through LAGB is able to prevent type 2 diabetes and hypertension for at least 4 years.

Acknowledgments— This research was supported by Grant FIRST 2002 from the Università degli Studi di Milano, the Ministero della Salute (grant RF-199/02), and the Ministero dell'Università e della Ricerca Scientifica e Tecnologica 2002 (grant 2002064582-003) to A.E.P. and from the Ministero della Salute (grant RF-02/224) to F.F.

We thank Tara Zoll Folli for her assistance in editing the manuscript.

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