



Effects of surgically induced weight loss on urinary bladder pressure, sagittal abdominal diameter and obesity co-morbidity

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OBJECTIVE: Evaluate the effects of surgically induced weight loss on intra-abdominal pressure at one year, reflected in urinary bladder pressure, central obesity, measured by sagittal abdominal diameter and obesity co-morbidity.

DESIGN: Prospective, non-randomized trial.

SETTING: University Hospital, Operating Room, In-patient, Outpatient Clinics.

SUBJECTS: Gastric bypass in 15 severely obese patients.

MEASUREMENTS: Patients underwent pre-operative assessment of weight, body mass index (BMI), co-morbid history, urinary bladder pressure and sagittal abdominal diameter. Patients were reassessed one year after gastric bypass with repeat measurement of weight, bladder pressure, and sagittal abdominal diameter and assessment of co-morbidity.

RESULTS: There were significant ($P < 0.001$) decreases in weight (140 ± 8 – 87 ± 6 kg), BMI (52 ± 3 – 33 ± 2 kg/m²), sagittal abdominal diameter (32 ± 1 – 20 ± 2 cm), urinary bladder pressure (17 ± 2 – 10 ± 1 cm H₂O) and obesity related problems per patient (2.9 ± 0.4 – 1 ± 0.2) one year after gastric bypass, with $69 \pm 4\%$ loss of excess weight.

CONCLUSIONS: Increased sagittal abdominal diameter is associated with increased intra-abdominal pressure which contributes to obesity related co-morbidity. Weight loss following gastric bypass decreases abdominal pressure, sagittal abdominal diameter and obesity co-morbidity.

Keywords: central obesity; gastric bypass; surgery; sagittal abdominal diameter; urinary bladder pressure; obesity co-morbidity

Introduction

There is general agreement that obesity is associated with increased morbidity and mortality^{1,2} and that this increased health risk is positively correlated with regional fat distribution and not simply the degree of obesity.^{3–5} The metabolic activity of visceral adipose tissue appears responsible for risk factors, collectively known as ‘syndrome X’, such as insulin resistance, hyperinsulinemia and diabetes, hypertension, and altered lipid metabolism.^{6–8} In a previous study, we found that central obesity is also associated with increased intra-abdominal pressure, as measured by urinary bladder pressure, and this is associated with a number of co-morbid problems. We hypothesized that several of these are probably due to increased intra-abdominal pressure and include hypoventilation, urinary incontinence, venous stasis, gastroesophageal reflux and incisional hernia, and hypertension.⁹ This study also supported data from other reports^{10–12} that sagittal abdominal diameter is a more reliable indica-

tor of central obesity than waist to hip ratio (WHR). The current study was designed to evaluate the effects of surgically induced weight loss on sagittal abdominal diameter, abdominal pressure as estimated from urinary bladder pressure and obesity co-morbidity.

Methods

Patient population

15 morbidly obese patients (body mass index (BMI) ≥ 35 kg/m²) of 84 previously reported patients⁹ undergoing surgery for morbid obesity at the Medical College of Virginia, Virginia Commonwealth University, were entered into the study. The study was approved by the Committee on the Conduct of Human Research. Patient weight, height, BMI and ideal body weight according to the 1983 Metropolitan Life Insurance Company actuarial tables¹³ were determined pre-operatively and informed consent obtained.

Abdominal diameter and bladder pressure

At the time of surgery, patients were placed supine on the operating table, which was leveled with a spirit level. Following induction of anesthesia, the patient was paralyzed and intubated. The sagittal abdominal

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diameter was measured as the distance between the table and the apex of the abdomen using a spirit level as the second reference point. Intra-abdominal pressure was estimated from the urinary bladder pressure. The bladder was catheterized and allowed to empty; a water manometer was connected by a three way stopcock to a rubber port on the urine collection tube with an 18 gage needle. The collection tube was then clamped just beyond its connection to the bladder catheter and 100 ml of 0.9% sterile saline instilled into the bladder. The water manometer, zeroed to the urethral meatus in women and the base of the penis in men, was then opened to the patient and the bladder pressure recorded in cm of water with the patient supine and the bed or operating room table leveled with a spirit level. Sterile technique was used at each step. Pre- and post-weight-loss pressures were measured once each. Respiratory oscillations of the fluid in the water manometer were noted in all patients. Both bladder pressure and sagittal abdominal diameter were recorded at end-expiration.

Obesity co-morbidity

There were 12 associated medical problems seen in this patient population which were subdivided into probable abdominal pressure related co-morbidity and non-pressure related co-morbidity. Probable abdominal pressure related co-morbidity included urinary incontinence, gastroesophageal reflux, lower limb venous insufficiency, obesity hypoventilation, systemic hypertension and congestive heart failure. Non-pressure related co-morbidity included degenerative joint disease, back pain, sleep apnea, Type II diabetes and gallstones.

Stress incontinence, gastroesophageal reflux and chronic joint or back pains were primarily diagnosed from the patient's reported symptoms. Five of the 15 patients with gastroesophageal reflux symptoms underwent 24 h pH evaluation and it was abnormal in each. The diagnosis of lower limb venous stasis disease required chronic edema with either pretibial bronze skin discoloration or an ulcer. Abdominal hernias were diagnosed on pre-operative physical examination or at the time of abdominal exploration. A diagnosis of hypertension was made with a persistently raised blood pressure (diastolic > 12 kPa and/or systolic $> 21/3$ kPa, using an extra-wide blood pressure cuff and phase IV Korotkoff sounds) or current anti-hypertensive treatment and Type II diabetes mellitus by a history of current diabetic medication (oral hypoglycaemic agents or insulin) or an abnormal fasting glucose (> 5.7 mmol/l). Sleep apnea syndrome was diagnosed by pre-operative sleep polysomnography (sleep apnea index (SAI), = # apneas + hypopneas/h of sleep, ≥ 5) in patients with a history of daytime somnolence, loud snoring and restless sleep. Obesity hypoventilation syndrome was diagnosed in severely dyspneic patients if the patient's PaO₂ was 7.3 kPa or PaCO₂ ≥ 6.27 kPa. Right heart

catheterization was performed in these patients and, if their pulmonary artery pressure was ≥ 5.3 kPa, they underwent prophylactic insertion of a Greenfield[®] vena caval filter at the time of gastric bypass.¹⁴ Congestive heart failure was diagnosed if the pulmonary capillary wedge pressure was ≥ 3.3 kPa and they responded to diuretic treatment. The presence of gallstones was noted at the time of gastric bypass by direct examination or intra-operative gallbladder ultrasonography. Post-weight-reduction gallstones were diagnosed with symptom directed ultrasonography.

Surgical intervention

13 patients underwent a 'proximal' 45 cm Roux-en-Y gastric bypass.¹⁵ In the latter phase of the study, two 'superobese' patients with pre-operative BMI's of ≥ 50 kg/m² underwent a 'long-limb' 150 cm Roux-en-Y gastric bypass.¹⁶ Prior to that change, there were seven 'superobese' patients who had a 'proximal' gastric bypass.

Postoperative evaluation

Fifteen of the patients who returned at one year follow-up after surgery, underwent repeat measurements of urinary bladder pressure and sagittal abdominal diameter, either in anesthetized patients who required surgery for incisional hernia or gallbladder disease or without anesthesia in the outpatient clinic. Postoperative co-morbidity was assessed as in the pre-operative patients. The patients were considered to no longer have: Type II diabetes mellitus if they had a normal fasting blood sugar (< 3.4 mmol/l) and no longer required either insulin or an oral hypoglycaemic agent; hypertension if their blood pressure was $< 18.7/11.3$ kPa without any anti-hypertensive medications or diuretics; gastroesophageal reflux if they had no heart burn and no longer required an acid reducing medication; sleep apnea syndrome if they no longer had daytime somnolence or nocturnal restlessness without the use of nasal continuous positive airway pressure (CPAP) or tracheostomy, or obesity hypoventilation syndrome if their PaO₂ was ≥ 8 kPa and PaCO₂ ≤ 5.6 kPa on room air.

Statistical analyses

Data are expressed as means \pm s.e.m. Comparison of pre- and postoperative means were performed using Student's paired *t*-tests. Significance was accepted with a *P* value of < 0.05 .

Results

Postoperative bladder pressure

Four of the first 19 patients of the previously reported 84 patients⁹ seen in follow-up at one year refused

urinary bladder catheterization. Weight, height and co-morbid history were obtained in the remaining 15 patients (2 males, 13 females). Sagittal abdominal diameter and urinary bladder pressure were measured in 12 patients without anesthesia; 3 patients were studied following anesthetic induction, paralysis and intubation for either incisional herniorrhaphy (two patients) or cholecystectomy (one patient). Mean pre-operative weight in the 15 follow-up patients was 140 ± 8 kg, which fell to 87 ± 6 kg at one year (Figure 1), a loss of $69 \pm 4\%$ of their excess weight ($P < 0.0001$). Weight loss was associated with a significant reduction in sagittal abdominal diameter from 32 ± 1 – 20 ± 2 cm, $P < 0.0001$ (Figure 2) and in urinary bladder pressure from 17 ± 2 – 10 ± 1.0 cm H₂O, $P < 0.001$ (Figure 3). In addition to the two patients who had urinary bladder pressures measured at the time of herniorrhaphy, five additional patients had incisional hernias at the time of bladder pressure measurement. Two of these seven patients had pre-operative incisional hernias. In the eight patients without pre- or postoperative incisional hernias, there were still significant ($P < 0.01$) decreases in weight (141 ± 10 – 90 ± 6 kg), sagittal abdominal diameter (30.6 ± 2 – 21.8 ± 2 cm) and urinary bladder pressure (17.9 ± 3 – 8.8 ± 1 cm H₂O) one year following surgically induced weight loss. All 15 patients demonstrated an improvement in co-morbidity (Table 1), with a mean reduction in obesity-related medical problems per patient from 2.9 ± 0.4 – 1 ± 0.2 ($P < 0.0001$). This fall in co-morbidity represented a reduction in non-pressure related factors and a complete resolution of all pressure related factors, except postoperative incisional hernia.

Discussion

All 15 patients in this study in whom urinary bladder pressures were measured before and one year after surgically induced weight loss, exhibited a decrease in sagittal abdominal diameter and all but one patient also had a reduced urinary bladder pressure. This was associated with a significant improvement in obesity co-morbidity. A potential source of error in these measurements could be the difference between measuring bladder pressures in awake vs anesthetized and paralyzed patients. Procedural discomfort may raise intra-abdominal pressure and hence bladder pressure, thus rendering the postoperative clinic measurements falsely high. This might introduce a potential negative bias in our results; therefore, significant positive conclusions should remain valid. As incisional hernias might lower intra-abdominal pressure and, therefore, urinary bladder pressure, the patients without pre- or postoperative incisional hernias were analyzed separately and were still found to have a significant decrease in urinary bladder pressure following surgically induced weight loss.

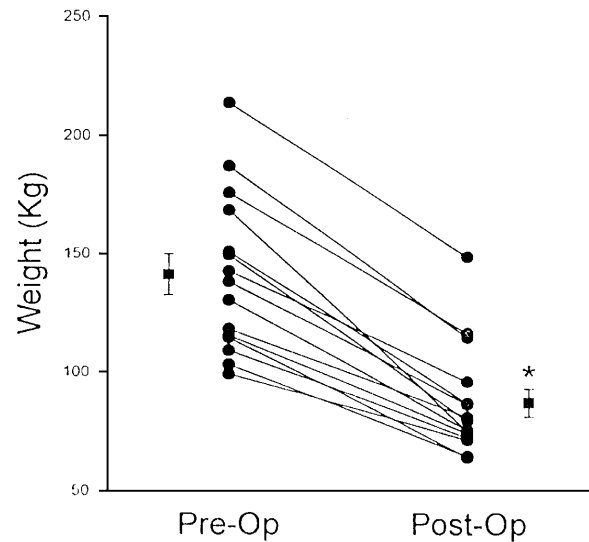


Figure 1 Effect of gastric bypass on weight in patients with pre- and one year post surgical measurement of urinary bladder pressure. ● = individual patient, ■ = mean \pm standard error of the mean, * = $P < 0.0001$.

Increased urinary bladder pressure and its relationship with increased intra-abdominal pressure has been well documented¹⁷ and has been used in postoperative surgical patients as an indication for re-exploration and abdominal decompression when the abdominal pressure is ≥ 20 cm H₂O to correct oliguria and respiratory failure.^{18–21} We have previously demonstrated an increased urinary bladder pressure and its association with stress and/or urge overflow urinary incontinence in the morbidly obese.²² In that study urinary bladder pressure was measured at 45°, sitting and standing; the latter produced a further increase in bladder pressure.²² This led to a much larger study which noted a significant correlation between urinary bladder pressure and sagittal abdominal diameter.⁹ A significantly higher urinary bladder pressure was also noted in patients with probable abdominal-pressure-related co-morbidity problems in contrast to those without pressure-related co-morbidity.⁹ Many of these patients had bladder pressures as high, or higher, than patients with an acute abdominal compartment syndrome, suggesting that severely obese patients with central obesity have a chronic abdominal compartment syndrome.

In the current study, all patients lost weight after gastric bypass with an average loss of $69 \pm 4\%$ of excess weight. This weight loss was associated with a marked reduction in both pressure related and non-related co-morbidity, except incisional hernia and need for cholecystectomy, with a significant ($P < 0.001$) reduced mean number of co-morbid factors per patient compared to pre-operative levels. Although the abdominal pressure-related morbidity factors chosen could be considered arbitrary, there are supporting data in pregnancy and cirrhotics with ascites, as well as morbidly obese women with urinary incontinence or individuals with obesity hypoventilation syndrome.^{22–28} In another study, we found a

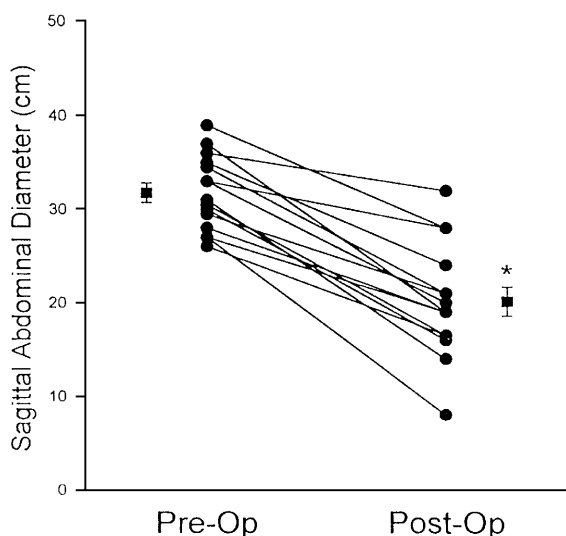


Figure 2 Sagittal abdominal diameter before and one year after surgically induced weight loss. ● = individual patient, ■ = mean ± standard error of the mean, * = $P < 0.0001$.

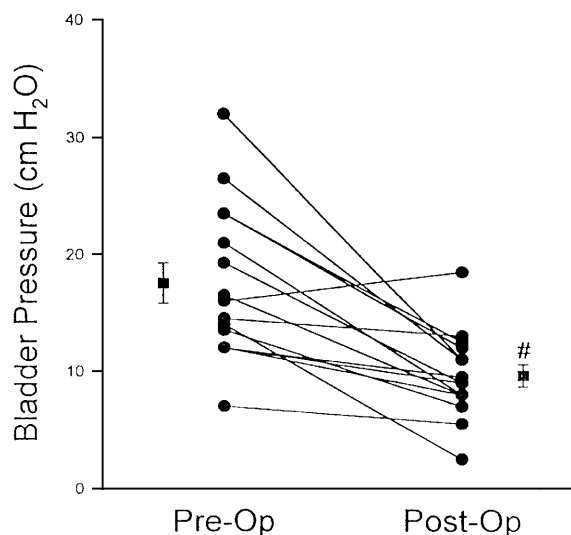


Figure 3 Urinary bladder pressure before and one year after surgically induced weight loss. ● = individual patient, ■ = mean ± standard error of the mean, * = $P < 0.001$.

Table 1 Obesity co-morbidity in 15 patients one year after gastric bypass

Co-morbid factor	Pre-Operative*	Postoperative*
Pressure related co-morbidity		
Gastroesophageal reflux	6	0
Stress overflow urinary incontinence	7	0
Abdominal hernia	2	7
Lower limb venous ulcer	2	0
Obesity hypoventilation syndrome	2	0
Systemic hypertension	10	2
Congestive heart failure	1	0
Non-pressure related co-morbidity		
Degenerative joint disease	4	2
Chronic low back pain	4	1
Sleep apnea syndrome	3	1
Gallstones/cholecystectomy	1	2
Type II diabetes mellitus	1	0
Total number of co-morbidity problems	43	15
Number of co-morbidity problems/patient	2.9 ± 0.4	1.0 ± 0.2

* $P < 0.001$ pre- vs postoperative.

significantly higher rate of incisional hernia (20% vs 4%, $P < 0.01$) following gastric bypass for severe obesity, than after a total abdominal colectomy, proctectomy and ileal pouch anal anastomosis for ulcerative colitis in mostly non-obese patients, many of whom were taking prednisone.²⁸ Much of the improvement in co-morbidity in the current study was based on subjective reports by the patients rather than objective data such as SAI from sleep polysomnography or acid reflux episodes from 24 h pH monitoring. This improvement may have been exaggerated by the patients' sense of euphoria over their significant weight loss and their desire to please the investigative team. To obtain objective follow-up data would be expensive and difficult in the current managed-care environment of the United States. Several studies have demonstrated improvement following surgically induced weight loss in conditions such as urinary incontinence,²² respiratory insufficiency,

including sleep apnea and obesity hypoventilation,¹⁴ Type II diabetes,³⁰ gastroesophageal reflux,³¹ idiopathic intracranial hypertension,³² hyperlipidemia,³⁴ female sexual hormone dysfunction,³⁵ hypertension^{36,37} and cardiac dysfunction.³⁸

We have hypothesized that hypertension and idiopathic intracranial hypertension are secondary to increased intra-abdominal pressure.⁹ To summarize, it is presumed that an increase in renal venous pressure and/or renal capsular pressure and/or a decrease in cardiac output, secondary to decreased venous return from an increased pleural pressure, individually or together lead to activation of the renin-angiotensin-aldosterone system with increased sodium and water reabsorption and vasoconstriction.^{39,40} Despite the compensated state with fluid retention and an increased cardiac output, patients with central obesity and hypertension have a decreased effective renal blood and plasma flow,⁴¹ a

phenomenon which is probably due to increased renal venous pressure and decreased renal perfusion. The increased pleural pressure impedes venous return from the brain, leading to an increased intracranial pressure.^{42,43}

The authors recognize that the data in this study do not prove causality between an increased intra-abdominal pressure and the presumed pressure-related co-morbidities. Our previous study found an increased sagittal abdominal pressure and urinary bladder pressure in severely obese individuals and hypothesized a relationship between these phenomena and obesity-related co-morbidity.⁹ The current study has found a decrease in sagittal abdominal pressure, urinary bladder pressure and obesity co-morbidity following surgically induced weight loss. We believe that these changes are causally related. We are in the process of developing an externally applied device which reduces increased intra-abdominal pressure and plan to use this device to treat severely obese patients with systemic hypertension, gastroesophageal reflux disease, obesity hypoventilation, venous stasis disease and pseudotumor cerebri. Results of these studies should provide further data to evaluate the proposed hypothesis that increased intra-abdominal pressure is responsible for these co-morbidities.

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