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SURGICAL ENDOSCOPY
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The impact of laparoscopy on bariatric surgery

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Abstract. The rising popularity of bariatric surgery over the past several years is attributable in part to the development of laparoscopic bariatric surgery. Morbidly obese patients have associated comorbid conditions that may predispose them to postoperative morbidity. The laparoscopic approach to bariatric surgery offers a minimally invasive option that reduces the physiologic stress and provides clinical benefits, as compared with the open approach. This review summarizes the impact of laparoscopic surgery on bariatric surgery, the various risk factors that could potentially predispose morbidly obese patients to postoperative morbidity, the fundamental differences between laparoscopic and open bariatric surgery, and the physiology of reduced tissue injury associated with laparoscopic bariatric surgery.

Key words: Laparoscopy — Morbid obesity — Bariatric surgery — Gastric bypass

Over the past four decades, many types of operations have been developed for the treatment of clinically severe obesity. Nonetheless, only three standards are currently in widespread use today. These procedures are the Roux-en-Y gastric bypass (GBP), adjustable gastric banding, and biliopancreatic diversion. Although bariatric surgery was developed in the late 1960s, it was not until the late 1990s that there was a significant growth in the number of bariatric operations performed in the United States. This rise in popularity is in part attributable to the development of the laparoscopic approach. Although the exact figure is difficult to ascertain, since the mid-1990s, Inamed (Santa Barbara, CA, USA) has sold more than 100,000 laparoscopic adjustable gastric bands worldwide [32]. There also has been an exponential increase in the number of Medicare and Medicaid patients receiving bariatric surgery (934 operations performed in 1998 and 3,424 operations performed in 2001) [53].

The growing enthusiasm for laparoscopic bariatric surgery is partly because of the high demand from the public for bariatric surgery and the increase in interest of the surgeons learning this complex laparoscopic operation. There has been a tremendous growth in the membership of the American Society for Bariatric Surgery and the attendance at its clinical program over the past several years. This phenomenon is similar to that experienced with laparoscopic cholecystectomy and laparoscopic Nissen fundoplication [46]. The number of these cases dramatically increased as the laparoscopic approach was adopted. For example, laparoscopic cholecystectomy surpassed open cholecystectomy only 4 years after the introduction of the laparoscopic method [46]. In essence, the public and medical physician acknowledged the benefits of laparoscopy and the improved risk to benefit ratio and surgery became a viable option.

Laparoscopic GBP and adjustable gastric banding both were developed in the early 1990s [7, 63]. Outcomes of laparoscopic GBP have been reported by multiple single institutional studies [23, 37, 47, 62]. These studies demonstrated a reduction in selected postoperative morbidity and mortality similar to that of the open approach. A single prospective randomized comparison of laparoscopic and open GBP has clearly demonstrated a significant reduction in postoperative pain, hospital stay, certain perioperative morbidity, and convalescence after laparoscopic GBP [37, 42]. However, the complexity of laparoscopic GBP for the morbidly obese patient has hindered the widespread transition of the procedure to common practices, compared with other laparoscopic procedures (cholecystectomy and Nissen fundoplication). A decade after the introduction of the laparoscopic method, laparoscopic GBP still has not surpassed open GBP as the most common treatment for morbid obesity.

In this review, we attempted to understand the various risk factors that could potentially predispose morbidly obese patients to postoperative morbidity, the

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Table 1. Comorbidities associated with obesity.

Increased risk of asthma
Increased risk of diabetes
Increased risk of stroke
Increased risk of heart failure
Increased risk of myocardial infarction
Increased risk of congestive heart failure
Increased risk of pulmonary embolism
Increased risk of deep venous thrombosis
Increased risk of thrombophlebitis
Increased risk of venous stasis ulcers
Increased risk of skin infections
Increased risk of all types of infections
Increased risk of obstructive sleep apnea
Increased risk of obesity hypoventilation syndrome
Idiopathic cranial hypertension
Increased risk for pulmonary hypertension
Increased risk of gallstones
Increased risk of arthritis
Increased risk of gout
Increased risk of dyslipidemia
Increased risk of reflux disease
Increased risk of infertility
Increased risk of miscarriage
Increased risk of polycystic ovarian syndrome
Increased risk of fetal abnormalities
Increased risk of perinatal infant mortality
Increased risk of depression
Increased risk of urinary incontinence
Increased risk of urinary tract infections
Increased risk for abdominal hernia
Increased risk of developing selected cancer
Increased risk of death
Increased risk of anesthetic-related death
Increased risk of adverse anesthetic-related respiratory outcomes

Table 2. Metabolic and physiologic risk factors associated with obesity

Cardiovascular Physiology
Increased intravascular volume [9]
Increased cardiac output [9]
Decreased total peripheral vascular resistance [9]
Eccentric left ventricular hypertrophy (LVH) [9]
Abnormal left ventricle filling [9]
Increased intraabdominal pressure [9]
Cardiovascular Metabolism
Increased levels of angiotensinogen [50]
Increased levels of renin [15]
Increased levels of angiotensin I [10]
Increased levels of angiotensin II [10]
Increased levels of aldosterone [20]
Increased levels of angiotensin-converting enzyme [22]
Increased levels of norepinephrine [29]
Haemostasis and Fibrinolytic Systems
Increased activation of Hageman factor [9]
Increased levels of fibrinogen [31]
Increased levels of factor VII [31]
Increased levels of factor VIII [31]
Increased levels of von Willebrand factor [31]
Increased levels of plasminogen activator inhibitor [31]
Increased RBC aggregation [58]
Immunologic
Expanded monocyte CD 14 receptor expression [12]
Expanded CD 14+/CD16+ monocyte subset [12]
Increased expression of leukocyte apoptotic receptors (CD95+ leukocytes) [11]
Decreased expression of adhesion receptor L-selectin (CD62L) [11]
Inflammatory
Increased levels of IL-1β [13]
Increased levels of IL-1RA [30]
Increased levels of IL-6 [8]
Decreased levels of IL-8 [4]
Increased levels of TNFα [24]
Increased levels TNFRA [25]
Increased levels of TGFβ [1]
Increase levels of C3 [52]
Increased levels of haptoglobin [52]
Increased levels of C-reactive protein [60]
Increased levels of serum amyloid A [15]

RBC, red blood cells; IL, interleukin; TNF, tumor necrosis factor; TGF, transforming growth factor

fundamental differences between laparoscopic and open bariatric surgery, and the physiology of reduced tissue injury associated with laparoscopic bariatric surgery.

Risk factors

Obesity increases the overall morbidity and mortality risk of an individual [6, 27, 54] (Table 1). As body mass index (BMI) increases, so does the likelihood of perioperative morbidity and mortality [17]. Specific conditions that may predispose bariatric surgical patients to a higher perioperative risk include preexisting comorbidities such as diabetes, obesity-hypoventilation syndrome, chronic obstructive pulmonary disease, hypertension, coronary artery disease, and sleep apnea. Many of these obesity-related comorbidities result from a wide range of metabolic and physiologic risk factors present in the obese, the most prominent being inflammatory (Table 2). This phenomenon was best established by Visser et al. [60] in 1999 and Ridker et al. [44] in 2000. These authors established a relation between C-reactive protein (an acute-phase reactant) and BMI using large cohort of patients. Ridker et al. [45] also demonstrated a positive association between C-reactive protein levels in obesity and the incidence of cardiovascular events. This finding is important because one of the causes for perioperative death in the morbidly obese is cardiovascular related. The presence of chronic inflammation in the morbidly obese affects a wide range of biologic processes that could have an impact on the perioperative outcomes of surgery. In addition, the increased intraabdominal pressure in the obese has been proposed as a link to many obesity comorbid conditions [55].

One of the most pressing issues that bariatric surgeons face is the threat of deep venous thrombosis (DVT) and pulmonary embolus. Most bariatric surgeons take multiple measures to prevent these dreaded complications (Table 3). There are some data to support the evidence that obese individuals have an increased risk of DVT and pulmonary embolus [5, 31, 58]. Many of these metabolic changes are related to chronic inflammation. Mertens et al. [31] recently showed how the increase in fat mass is associated with elevated levels of fibrinogen, factor VII, factor VIII, von Willebrands factor and plasminogen activator inhibitor (PAI). Other studies also have shown increased red blood cell adhesive/aggregation properties in obesity [5, 58].

Table 3. Steps to decrease the risk for development of postoperative deep venous thrombosis and pulmonary embolism

1. Sequential compression devices placed before induction of anesthesia
2. Prophylactic anticoagulation of patient 2 h before induction of anesthesia
3. Prophylactic anticoagulation of patient postoperatively
4. Early postoperative ambulation

Infection is another cause for morbidity and mortality in obesity. This, along with the increased incidence of cancer, could possibly be related to inflammatory and immunologic changes in the obese. These changes limit the ability of leukocytes to migrate to sites of infection and increase the chances of cell death. Impaired migration occurs with the downregulation of L-selectin on leukocytes. L-selectin is a cell surface receptor necessary for leukocyte migration [11, 14]. Increased risk of cell death (apoptosis) occurs with the increased expression of CD95, the Fas antigen. This cell surface receptor, belonging to the tumor necrosis family, is upregulated in obesity and predisposes leukocytes to programmed cell death when inflammatory insults are present. These changes impair the immunologic systems of the obese and leave them less able to fight infection [12].

Diabetes and its management are an important part of the care for the morbidly obese. The development of type 2 diabetes in morbid obesity is directly correlated with the amount of excess adipose tissue and its ability to secrete tumor necrosis factor (TNF-α), which causes insulin resistance. There is much evidence to support the aforementioned hypothesis because both medication and dietary modifications improve insulin sensitivity, which coincides with a decrease in levels of TNF-α and weight loss [19, 34]. The exact mechanism is related to TNF-α receptor expression, as obese mouse models lacking either TNF-α or its receptors almost totally ameliorate insulin resistance [56, 57, 59]. The elevated levels of TNF in obesity could potentially compromise many different systems other than just glucose metabolism.

Fundamental differences between laparoscopic and open bariatric surgery

It is important to understand the differences between open and laparoscopic surgery because the differences in technique between laparoscopic and open bariatric surgery may account for the different biologic responses in the two approaches. The major differences between open and laparoscopic bariatric surgery are the method of access, the carbon dioxide (CO_2) pneumoperitoneum, and the degree of tissue injury (Table 4). Reduced tissue injury probably is the primary factor accounting for the beneficial effects of laparoscopic bariatric surgery, and the use of CO_2 pneumoperitoneum is the factor accounting for the adverse physiologic changes during laparoscopic surgery [41]. The detrimental effects of CO_2 pneumoperitoneum occur transiently during the intraoperative period, whereas the beneficial effects of reduced tissue injury affects the period from injury to recovery.

Physiology of reduced tissue injury associated with laparoscopic bariatric surgery

The biologic response to surgical injury is dependent on the magnitude of the surgical insult and the host's ability to recover. Open bariatric surgery is associated with significant surgical insult and often is followed by a period of metabolic, acute-phase, cytokine, and catabolic responses [61]. The primary goal in minimally invasive surgery is to reduce the tissue injury and subsequently reduce the maladaptive host responses to injury. Laparoscopic GBP accomplishes the same technical and anatomic objective as open GBP, but avoids a large abdominal incision and reduces the abdominal viscera trauma. By eliminating the large abdominal wound (> 10 cm) and reducing tissue injury, laparoscopic GBP has been postulated to lessen the extent of operative injury and result in an attenuated systemic stress response.

The degree of tissue injury

Open Roux-en-Y GBP is a major upper abdominal operation requiring a large abdominal incision, extensive surgical dissection, and significant bowel and viscera manipulation. The tissue injury during open GBP results in postoperative tissue edema and fluid retention (third-space fluid) of the surgical wounds [61]. The degree of third-space fluid accumulation can be quantified by measurement of the intraabdominal pressure (IAP) because the abdominal cavity is a single cavity [26]. The degree of increase in IAP is proportional to the extent of intraabdominal tissue dissection, bowel manipulation, and surgical abdominal wall trauma [38, 61]. Compounding factors such as intraperitoneal bleeding and bowel distention also can further increase the postoperative IAP. Kron et al. [26] measured the IAP of patients undergoing elective surgical operations such as colon resection, vascular reconstruction, and lung resection. The IAP within the first 24 h after surgery ranged from 3 to 13 mm Hg with the use of an intravesical method.

Laparoscopic GBP reduces the length of the abdominal incision, minimizes tissue trauma from abdominal wall retraction, and reduces manipulation of the abdominal viscera. Nguyen et al. [38] demonstrated that laparoscopic GBP resulted in less surgical trauma than open GBP. Elevated IAP occurred immediately after both laparoscopic and open GBP. However, IAP was significantly lower on the first postoperative day after laparoscopic GBP than after open GBP (12.5 vs 17.2 cm H_2O, respectively). The results reported by these authors support the notion that laparoscopic GBP is associated with a lower degree of tissue injury.

Table 4. Factors that may account for differences in physiologic responses between open and laparoscopic bariatric surgery

Factor	Open bariatric surgery	Laparoscopic briatric surgery
Anesthesia	Equivalent	Equivalent
Patient position	Supine/reverse Trendelenburg	Supine/reverse Trendelenburg
Abdominal incision	Upper midline incision through skin, fascia	Two 12-mm and three 5-mm trocar incisions
Retraction	Abdominal wall retractor (i.e., Bookwalter)	CO_2 pneumoperitoneum and gravity
Intraabdominal visceral retraction/manipulation	Significant (using hands, metal retractors, and sponge)	Minimal
Heat loss	Moderate	Moderate

CO_2, Carbon dioxide

Table 5. Systemic stress response after laparoscopic and open gastric bypass (GBP)

Levels	Outcome
TNF-α	No differences between laparoscopic and open GBP
IL-8	No differences between laparoscopic and open GBP
Dopamine	No differences between laparoscopic and open GBP
Epinephrine	No differences between laparoscopic and open GBP
Insulin	No differences between laparoscopic and open GBP
Glucose	No differences between laparoscopic and open GBP
Cortisol	No differences between laparoscopic and open GBP
Norepinephrine	Significantly higher in open GBP 24 h postoperatively
ACTH	Significantly higher in open GBP 1 h postoperatively
CRP	Significantly higher in open GBP 24 h postoperatively
IL-6	Significantly higher in open GBP 1, 48, and 72 h postoperatively

TNF, tumor necrosis factor-α; IL-8, interleukin-8; ACTH, adrenocorticotropic hormone; CRP, C-reactive protein; IL-6, interleukin-6

Metabolic responses to injury

The extent, degree, and duration of the metabolic changes that follow tissue injury depend on the severity of that injury. Specifically, findings have shown releases of catecholamine, glucose, adrenocorticotropic hormone (ACTH), C-reactive protein, and interleukin-6 to be proportional to the severity of injury [49, 61]. Peak levels of cortisol, however, are not a reliable measure for the extent of injury because the duration in the rise of cortisol levels predicts a major injury or sepsis. The neuroendocrine and cytokine responses to injury have been evaluated in patients who underwent either open or laparoscopic GBP (Table 5). Increases in ACTH and acute-phase reactant, such as C-reactive protein, were significantly less and returned to baseline quicker after laparoscopic GBP than after open GBP [36]. Findings have shown that indicators of cytokine response, such as levels of interleukin-6, are lower after laparoscopic GBP than after open GBP [36].

Pulmonary function after surgical injury

Impairment of pulmonary function after major abdominal surgery is well documented and may last as long as 10 days after the operation [6]. The changes in pulmonary function after laparoscopic surgery have been extensively studied, particularly after cholecystectomy and colectomy [33, 48, 51]. Schauer et al. [48] compared the postoperative pulmonary function of 40 patients who underwent laparoscopic and open cholecystectomy. On postoperative day 1, spirometric parameters (forced vital capacity [FVC], forced expiratory volume at 1s [FEV_1], forced expiratory volume at midexpiratory-phase $FEV_{25–75\%}$, and peak expiratory flow [PEF]) in the laparoscopic group decreased respectively, to 79%, 76%, 68%, and 76% of their preoperative levels, as compared with 49%, 44%, 34%, and 38% in the open cholecystectomy group. In addition, pulmonary complications (atelectasis and hypoxemia) were less frequent after laparoscopic cholecystectomy than after open cholecystectomy [48]. Nguyen et al. [39] demonstrated that pulmonary function was less impaired after laparoscopic GBP than after open GBP (Table 6). All respiratory flow parameters (FVC, FEV_1, $FEV_{25–75\%}$, and PEF) were decreased in both the laparoscopic and open GBP groups on postoperative day 1. However, the FVC, FEV_1, $FEV_{25–75\%}$, and PEF levels were higher after laparoscopic GBP than after open GBP by 36%, 38%, 31%, and 25%, respectively. All of the respiratory flow parameters returned to their preoperative values by postoperative day 7 in the laparoscopic GBP group, but only PEF had returned to preoperative values in the open GBP group by that time.

The lower levels of postoperative pain after laparoscopic GBP likely played a role in preserving postoperative pulmonary function. Postoperative pain contributes to chest wall splinting, tachypnea, and shallow breathing that can lead to atelectasis. The frequency of pulmonary segmental atelectasis on postop-

Table 6. Changes in postoperative pulmonary function, atelectasis, and postoperative pain between laparoscopic and open gastric bypass (GBP)

Factors	Open GBP	Laparoscopic GBP
FVC	39% of preoperative values	53% of preoperative values
FEV_1	39% of preoperative values	54% of preoperative values
$FEV_{25–75\%}$	41% of preoperative values	53% of preoperative values
PEF	41% of preoperative values	52% of preoperative values
Hypoxemia	76% of patients required supplemental oxygen	31% of patients required supplemental oxygen
MSO_4 consumption	76 mg on POD 1	46 mg on POD1
VAS	Higher VAS score than with laparoscopic GBP	

FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; $FEV_{25–75\%}$, forced expiratory volume at midexpiratory phase; PEF, peak expiratory flow; MOS_4, morphine sulfate consumption; POD, postoperative day; VAS, visual analog scale

Table 7. Laparoscopic bariatric surgery and postoperative venous thrombosis

Factor	Consequence
Pneumoperitoneum (15 mmHg)	Promotes venous stasis of the lower extremity. May promote venous distention and consequent endothelial damage
Reverse Trendelenburg position	Promotes venous stasis of the lower extremity
Increased operative time	Increases effect of all operative factors that may promote thrombosis
Increased postoperative mobility	Reduces venous stasis
Attenuated hypercoagulable response	May reduce associated risk of thrombosis

erative day 1 is reported to be lower for patients who undergo laparoscopic GBP than those who undergo open GBP [40].

Thrombosis and coagulation after laparoscopic bariatric surgery

Deep venous thrombosis and pulmonary embolism contribute significantly to the perioperative morbidity and mortality of patients undergoing bariatric surgery. The reported incidence of pulmonary embolism for patients undergoing open GBP with prophylaxis has ranged from 0.4% to 3% [2, 3, 18, 21, 28]. Schauer et al. [47] reported a 0.73% incidence of venous thromboembolism in a prospective series of 275 patients who underwent laparoscopic GBP. Wittgrove and Clark [62] reported no thromboembolic complications in a series of 500 laparoscopic GBP patients, and Higa et al. [23] reported a 0.2% incidence of DVT as well as a 0.3% incidence of pulmonary embolism in 1,040 laparoscopic GBP patients. To date, the evidence is inconclusive as to the relative risk of postoperative thromboembolism after laparoscopic GBP, as compared with open GBP. All elements of Virchow's triad (venous stasis, hypercoagulability, and endothelial injury) may influence the risk of postoperative DVT during laparoscopic GBP.

Several factors specific to the laparoscopic method may increase the risk of DVT associated with laparoscopic GBP (Table 7). The increased intraabdominal pressure during pneumoperitoneum can result in reduced venous flow in the lower extremities. Nguyen et al. [35] reported a significant reduction in peak systolic velocity of the femoral vein during laparoscopic GBP with pneumoperitoneum at 15 mmHg. Intermittent sequential pneumatic compression partially reverses the reduction in peak flow velocity of the femoral vein. In addition, the reverse Trendelenburg position during laparoscopic GBP has been shown to promote intraoperative venous stasis, and the operative time is frequently longer for laparoscopic procedures, particularly during the surgeon's learning curve.

Table 8. Changes in coagulation and fibrinolysis after laparoscopic and open gastric bypass (GBP)

Levels	Outcomes
Protein C	Significantly greater in laparoscopic GBP at 72 h
Plasminogen	No differences
Fibrinogen	No differences
Prothrombin fragment	No differences
D-Dimer	Significantly lower in laparoscopic GBP at 1 and 24 h
Antithrombin III	Significantly greater in laparoscopic GBP at 1 h

In contrast, several factors specific to laparoscopic GBP may decrease the risk of DVT. The degree of coagulation cascade (hypercoagulable state) activation has been demonstrated to be attenuated after laparoscopic GBP, as compared with open GBP [40]. Nguyen et al. [40] demonstrated that the combination of increased thrombin production and reduced antithrombotic activity seen after laparoscopic GBP confirms its hypercoagulable state and hence the risk for postoperative DVT (Table 8). However, the reduction of antithrombotic parameters (antithrombin III and protein C) and the increase in D-dimer levels were less at specific time points after laparoscopic GBP than after open GBP [40]. In addition, the hypercoagulable state after an operation may be regulated by various cytokines, such as interleukin-6. Plasma concentrations of interleukin-6 are reported to be significantly lower after laparoscopic GBP than after open GBP [36]. The enhanced mobility

after laparoscopic GBP also may reduce venous stasis and the risk of thrombosis [46].

Summary

Laparoscopic bariatric surgery has made a significant impact on the field of bariatric surgery. The growing enthusiasm for laparoscopic bariatric surgery is based on the high demand for the procedure by the public and an increase in the interest of surgeons for performing this technically challenging operation. Minimally invasive approaches to bariatric surgery offer clinical advantages over open surgery. The benefits of laparoscopic bariatric surgery is particularly important for the morbidly obese because these patients have significant existing comorbidities that could potentially predispose them to postoperative morbidity. Currently, there are evidenced-based data to support the concept that as compared with open bariatric surgery, laparoscopic bariatric surgery results in a lower degree of tissue injury, attenuated metabolic and cytokine responses, improved pulmonary function, and an attenuated systemic coagulation and fibrinolysis response.

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