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SURGICAL ENDOSCOPY
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Cardiac function during laparoscopic vs open gastric bypass

A randomized comparison

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Abstract
Background: Hypercarbia and increased intraabdominal pressure during prolonged pneumoperitoneum can adversely affect cardiac function. This study compared the intraoperative hemodynamics of morbidly obese patients during laparoscopic and open gastric bypass (GBP).
Methods: Fifty-one patients with a body mass index (BMI) of 40–60 kg/m^2 were randomly allocated to undergo laparoscopic ($n = 25$) or open ($n = 26$) GBP. Cardiac output (CO), mean pulmonary artery pressure (MPAP), pulmonary artery wedge pressure (PAWP), central venous pressure (CVP), heart rate (HR), and mean arterial pressure (MAP) were recorded at baseline, intraoperatively at 30-min intervals, and in the recovery room. Systemic vascular resistance (SVR) and stroke volume (SV) were also calculated.
Results: The two groups were similar in terms of age, weight, and BMI. Operative time was longer in the laparoscopic than in the open group ($p < 0.05$). The HR and MAP increased significantly from baseline intraoperatively, but there was no significant difference between the two groups. In the laparoscopic group, CO was unchanged after insufflation, but it increased by 5.3% at 2.5 h compared to baseline and by 43% compared to baseline in the recovery room. In contrast, during open GBP, CO increased significantly by 25% after surgical incision and remained elevated throughout the operation. CO was higher during open GBP than during laparoscopic GBP at 0.5 h and at 1 h after surgical incision ($p < 0.05$). During laparoscopic GBP, CVP, MPAP, and SVR increased transiently and PAWP remained unchanged. During open GBP, CVP, MPAP, and PAWP decreased transiently and SVR remained unchanged. There was no significant difference in the amount of intraoperative fluid administered during laparoscopic (5.5 ± 1.6 L) and open (5.6 ± 1.7 L) GBP.
Conclusion: Prolonged pneumoperitoneum during laparoscopic gastric bypass does not impair cardiac function and is well tolerated by morbidly obese patients.

Key words: Hemodynamics — Obesity — Gastric bypass — Laparoscopy — Cardiac function

Laparoscopic Roux-en-Y gastric bypass (GBP) has become a routine surgical procedure for the treatment of morbid obesity. Despite its acceptance, there remains concern about the potential adverse effects of prolonged (>3 h) carbon dioxide (CO_2) pneumoperitoneum on cardiovascular function. Pneumoperitoneum is associated with systemic absorption of CO_2 that can lead to hypercarbia and acidosis [5]. In addition, increased intraabdominal pressure and reverse Trendelenburg position during the laparoscopic operation may impede venous return and decrease cardiac function [1, 7, 10].

Several investigators have reported depressed cardiac function during short laparoscopic operations such as laparoscopic cholecystectomy [1, 7, 10, 12]. In addition, others have observed that a 15 mmHg pneumoperitoneum appears to be the threshold for a decrease in cardiac output [2, 3, 6, 11]. In an animal study, Kaklamanos et al. [8] also found that prolonged pneumoperitoneum negatively affected cardiac output. Given that laparoscopic GBP requires prolonged CO_2 pneumoperitoneum [14], the negative effect of pneumoperitoneum on cardiac function may be augmented during laparoscopic GBP. This physiologic derangement has been attributed to the sympathetic effects of hypercarbia, hypovolemia, the mechanical effects of increased intraabdominal pressure, intraoperative body positioning, or a combination of these factors.

In this study, we compared the perioperative cardiac functions of patients who underwent laparoscopic or open GBP. To minimize the effects of systemic hypercarbia and

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hypovolemia on cardiac function, we specifically monitored and maintained a normal systemic level of CO_2 during laparoscopic GBP (measured by arterial blood gas) and maintained an intraoperative euvolemic state in both groups (monitored by cardiac filling pressures).

Patients and methods

The study protocol was approved by the Institutional Review Board of the University of California–Davis Medical Center. All patients being evaluated for the surgical treatment of morbid obesity were considered for entry into the trial. Patients were considered eligible if they had a body mass index (BMI, calculated as weight in kilograms divided by the square of height in meters) of 40–60, were younger than 60 years, and had failed at previous nonsurgical attempts at weight loss. Patients who had previous obesity or gastric surgery, a large abdominal ventral hernia, a history of myocardial infarction, or severe coronary artery disease were excluded. After written informed consent was obtained, the patients were randomly assigned to undergo either laparoscopic or open GBP. Patients were informed of their treatment group during their preoperative clinic visit. Randomization was performed by using sealed envelopes that were stratified according to BMI of 40–49 or 50–60. Demographic data, BMI, operative time, operative hemodynamics, and amount of intraoperative fluid administered were recorded.

Anesthetic technique

Anesthetic management was performed similarly for both groups using a standardized protocol. Patients were premedicated with intravenous (IV) midazolam (1–2 mg) in the preoperative holding area. Standard physiologic monitors were placed, including electrocardiographic leads, pulse oximeter, and an automatic blood pressure cuff. Additional midazolam and fentanyl (3 mcg/kg) were given as clinically indicated. General anesthesia was induced with IV sodium pentothal, followed by IV succinylcholine to facilitate endotracheal tube placement. General anesthesia was maintained with incremental IV boluses of fentanyl ($\leq$10 mcg/kg), isoflurane (0.5–1.5% concentration), and incremental doses of vecuronium or pancuronium as needed. Muscle relaxation was monitored with a neuromuscular stimulator (Digistim II; Neuro Technology, Houston, TX, USA). Patients were ventilated with an F_iO_2 of 0.5 using a constant-flow time-cycled ventilator. Minute ventilation was adjusted to maintain an end-tidal CO_2 of 35–40 mmHg.

Intraoperative fluid loss was replaced with crystalloid solution (lactated Ringer's or normal saline). Fluid deficits from preoperative fasting were calculated from actual body weight and replaced during the first 3 h of surgery. Intraoperative maintenance fluid requirements were estimated at 10 mL/kg of ideal body weight and adjusted as clinically indicated.

Operative technique

Thigh length anti-embolic stockings (TED; Kendall Healthcare Products Co, Mansfield, MA, USA), and thigh-length pneumatic sequential compression sleeves (SCD; Kendall) were placed on both lower extremities for DVT prophylaxis in both groups. In both groups, a 15- to 20-mL transected gastric pouch was created, a 75-cm jejunal Roux limb was created for patients with a BMI of 40–49, and a 150-cm jejunal Roux limb was created for patients with a BMI of 50–60. A stapled gastrojejunostomy anastomosis was performed in both groups. Laparoscopic GBP was performed through five abdominal trocars; open GBP was performed through an upper midline incision. Our technique of laparoscopic GBP has been described previously [14]. Intraabdominal pressure was maintained at 15 mmHg during pneumoperitoneum.

Study protocol

The primary end point of this study was intraoperative cardiovascular hemodynamics. Radial and pulmonary artery catheters (UIP Swan-Ganz; Baxter Healthcare, Irvine, CA, USA) were placed after the induction of anesthesia for continuous hemodynamic monitoring. The radial arterial line was used for continuous monitoring of mean arterial pressure (MAP). Arterial blood gas samples were drawn at baseline and at 1-h intervals until completion of the operation.

Mean pulmonary arterial pressure (MPAP), central venous pressure (CVP), pulmonary arterial wedge pressure (PAWP), and cardiac output (CO) using the thermodilution technique were measured after the induction of general anesthesia, intraoperatively at 30-min intervals, and 30 min after arrival in the recovery room. The pressure transducer was located at the level of the right atrium during all measurements. Stroke volume (SV) was calculated as SV = CO/HR. Systemic vascular resistance (SVR) was calculated as SVR = [MAP– CVP] × 80/CO.

Postoperative care

At the completion of surgery, all patients were given neostigmine and glycopyrrolate to antagonize residual muscle relaxant effects before extubation. Morphine was used intraoperatively as clinically indicated to supplement postoperative analgesia. The radial arterial and pulmonary arterial catheters were removed in the recovery room. All patients were transferred to the surgical ward postoperatively unless they required ventilatory support or close observation in the intensive care unit. Patient-controlled analgesia using morphine was started in the recovery room once the patient became alert.

Statistical analysis

Data are presented as the mean ± standard deviation (SD). Demographic data were compared using a two-sample *t*-test or Fisher's exact test. Repeated-measures analysis of variance (ANOVA) was used to analyze all hemodynamic data. After the initial ANOVA, a series of stratified models were run to look for significant differences between groups at each time point using unpaired *t*-tests or significant differences from baseline within each group using paired *t*-tests. Statistical evaluations were performed using standardized software (Statview; SAS Institute Inc., Cary, NC, USA). A p value of < 0.05 was considered significant. All data were analyzed on an intention-to-treat basis. Laparoscopic GBP operations that were converted to open GBP were analyzed as laparoscopic operations.

Results

Patient demographic and operative data

Fifty-one patients were randomly allocated to undergo either laparoscopic ($n = 25$) or open ($n = 26$) GBP between May 1999 and April 2000. During this time, 10 eligible patients were not enrolled; nine of them specifically requested laparoscopic GBP, and one requested open GBP. One patient randomized to the open GBP group was excluded from the study after randomization; this patient had intraoperative hemorrhage that ultimately required splenectomy and did not undergo GBP. Two patients in the laparoscopic group required conversion to laparotomy—one for revision of the gastrojejunostomy anastomosis and the other for inability to insufflate the abdomen.

There was no significant difference between the two groups with respect to age, gender, American Society of Anesthesiology (ASA) classification, and preoperative BMI (Table 1). There was no significant difference between the groups in the amount of intraoperative fluid administered (5.57 ± 1.66 vs 5.66 ± 1.78 L, $p = 0.86$). The median length of hospital stay was 3 days after laparoscopic GBP and 5 days after open GBP.

Changes in HR, MAP, and arterial partial pressure of

Table 1. Characteristics and operative data of patients randomized to laparoscopic and open gastric bypass (GBP)

Characteristics	Laparoscopic GBP (n = 25)	Open GBP (n = 26)	p value
Gender (female: male)	24:1	23:3	NS[a]
Age (yr)	42 ± 8	46 ± 8	NS[b]
Preoperative BMI (kg/m^2)	48 ± 5	50 ± 6	NS[b]
ASA classification (1–5)	2.9 ± 0.3	2.8 ± 0.5	NS[b]
Intraoperative fluids administered (L)	5.57 ± 1.66	5.66 ± 1.78	NS[b]
Estimated blood loss (ml)	157 ± 98	390 ± 250	<0.001[c]
Operative time (h)	232 ± 51	205 ± 43	0.04[c]

BMI, body mass index; ASA, American Society of Anesthesiologists; NS, not significant
[a] Fisher's exact test
[b] 2-sample t-test
[c] Mann-Whitney U test

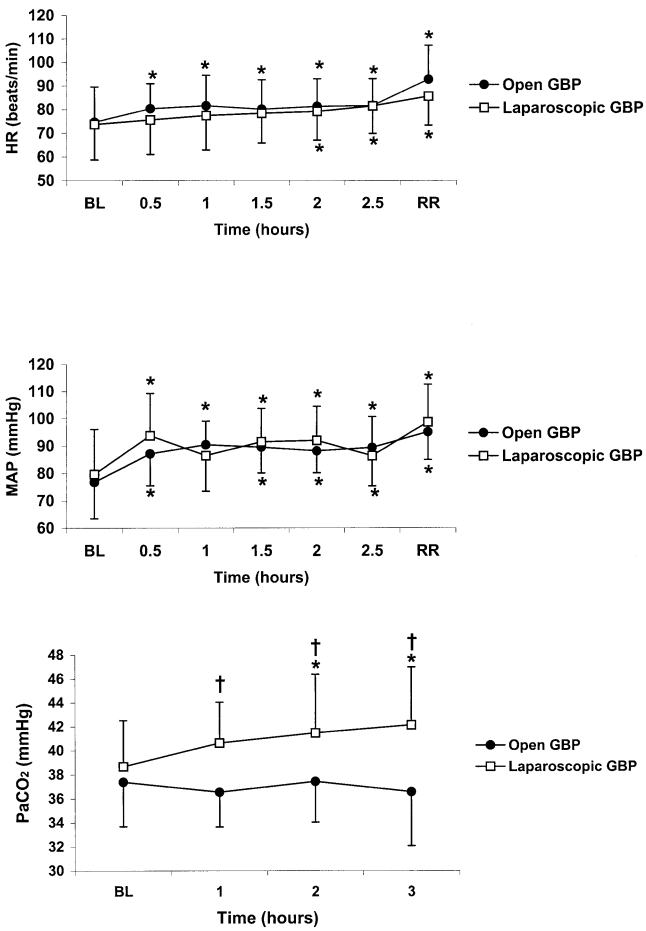

Fig. 1. Changes in heart rate (HR), mean arterial pressure (MAP), and mean arterial CO_2 levels ($PaCO_2$) after laparoscopic and open gastric bypass (GBP). BL = baseline; RR = recovery room; * $p < 0.05$ compared to baseline value (paired t-tests); † $p < 0.05$ compared to open GBP (unpaired t-tests).

CO_2 ($PaCO_2$) levels are presented in Fig. 1. During laparoscopic GBP, HR increased significantly from 73.7 ± 15.0 beats/min at baseline to 79.2 ± 12.1 beats/min at 2 h after abdominal insufflation ($p = 0.03$) and then increased further to 85.6 ± 12.2 beats/min in the recovery room ($p < 0.01$). During open GBP, HR increased immediately from 74.6 ± 14.8 beats/min at baseline to 80.4 ± 10.6 beats/min at 0.5 h ($p = 0.05$) and remained elevated throughout the operation. MAP increased immediately after the start of surgery during both laparoscopic (79.6 ± 16.4 mmHg at baseline vs 93.7 ± 15.5 mmHg at 0.5 h, $p < 0.01$) and open GBP (76.7 ± 13.2 mmHg at baseline vs 87.1 ± 11.6 mmHg at 0.5 h, $p < 0.01$) and remained elevated throughout the operation. There was no significant difference in MAP or HR between the two groups at any time. During laparoscopic GBP, $PaCO_2$ levels increased significantly from 38.6 ± 3.8 mmHg at baseline to 41.5 ± 4.9 mmHg at 2 h after insufflation ($p = 0.03$) and remained elevated throughout the operation. During open GBP, $PaCO_2$ levels remained unchanged from baseline. $PaCO_2$ levels were significantly higher during laparoscopic GBP than during open GBP at 1, 2, and 3 h after surgical incision ($p < 0.05$), but $PaCO_2$ levels were maintained at < 45 mmHg in the laparoscopic group throughout the operation.

Changes in CO, SV, and SVR are presented in Fig. 2. During laparoscopic GBP, CO levels were unchanged immediately after insufflation (7.2 ± 2.1 L/min at baseline vs 6.9 ± 2.0 L/min at 0.5 h, $p = 0.34$) but increased significantly to 7.6 ± 2.0 L/min at 2.5 h after insufflation ($p = 0.02$) and then increased to 10.3 ± 1.7 L/min in the recovery room ($p < 0.01$). During open GBP, CO increased significantly at the start of the operation from 6.8 ± 1.7 L/min at baseline to 8.5 ± 2.1 L/min at 0.5 h ($p < 0.01$) and remained elevated throughout the operation; they increased further to 10.3 ± 2.7 L/min in the recovery room ($p < 0.01$). CO levels were significantly greater during open GBP than during laparoscopic GBP at 0.5 h and at 1 h ($p < 0.05$). There was no significant difference in CO in the recovery room between groups. During laparoscopic GBP, SV transiently decreased after insufflation from 99.2 ± 21.4 mL/beat at baseline to 91.4 ± 21.5 mL/beat at 0.5 h ($p = 0.04$). SV levels immediately recovered to baseline value at 1.5 h after insufflation and increased significantly to 126.0 ± 39.2 mL/beat in the recovery room ($p = 0.05$). During open GBP, SV increased transiently from 92.3 ± 23.6 mL/beat at baseline to 106.2 ± 24.9 mL/beat at 0.5 h ($p = 0.01$) and increased again to 116.9 ± 27.4 mL/beat in the recovery room ($p < 0.01$). During laparoscopic GBP, SVR increased transiently from 727 ± 179 dynes • s • cm^5 at baseline to 975 ± 425 dynes • s • cm^5 at 0.5 h ($p < 0.01$). During open GBP, SVR remained unchanged throughout the operation.

Changes in CVP, PAWP, and MPAP are presented in

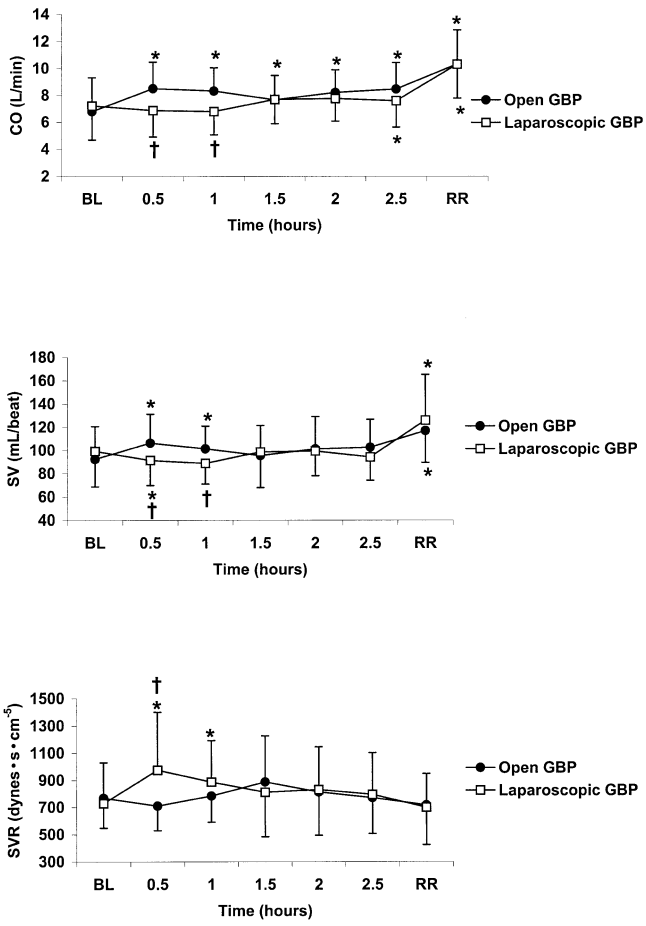

Fig. 2. Changes in cardiac output (CO), stroke volume (SV), and systemic vascular resistance (SVR) after laparoscopic and open gastric bypass (GBP). BL = baseline; RR = recovery room; * $p < 0.05$ compared to baseline value (paired *t*-tests); † $p < 0.05$ compared to open GBP (unpaired *t*-tests).

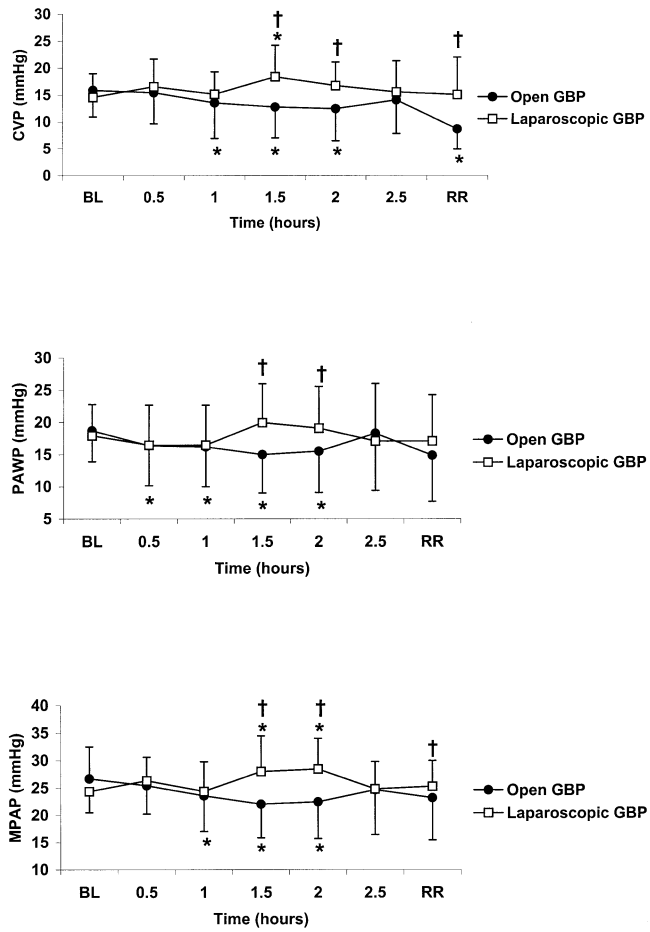

Fig. 3. Changes in central venous pressure (CVP), pulmonary artery wedge pressure (PAWP), and mean pulmonary artery pressure (MPAP) after laparoscopic and open gastric bypass (GBP). BL = baseline; RR = recovery room; * $p < 0.05$ compared to baseline value (paired *t*-tests); † $p < 0.05$ compared to open GBP (unpaired *t*-tests).

Fig. 3. During laparoscopic GBP, CVP increased significantly from 14.6 ± 3.6 mmHg at baseline to 18.4 ± 5.8 mmHg at 1.5 h ($p < 0.01$) and 16.7 ± 4.4 mmHg at 2 h after insufflation ($p < 0.01$); MPAP increased significantly from 24.3 ± 3.8 mmHg at baseline to 28.0 ± 6.5 mmHg at 1.5 h ($p = 0.01$); and PAWP remained unchanged from baseline values. During open GBP, CVP significantly decreased from 15.8 ± 3.1 mmHg at baseline to 12.7 ± 5.7 mmHg at 1.5 h ($p < 0.01$); PAWP decreased significantly from 18.7 ± 3.9 mmHg at baseline to 16.4 ± 6.1 mmHg at 0.5 h ($p = 0.04$); MPAP decreased significantly from 26.6 ± 5.8 mmHg at baseline to 23.6 ± 6.5 mmHg at 1 h after surgical incision ($p = 0.04$). CVP, PAWP, and MPAP were significantly greater at 1.5 h and at 2 h in the laparoscopic GBP group than in the open GBP group ($p < 0.05$).

Discussion

Many factors interact to determine intraoperative cardiac function. These factors include preload, afterload, contractility, heart rate, and myocardial compliance. Factors specific to CO_2 pneumoperitoneum (increased intraabdominal pressure, hypercarbia) or operative positioning (reverse Trendelenburg position) may also adversely affect cardiac function. Most clinical studies evaluating cardiac function during pneumoperitoneum have attributed the changes to increased intraabdominal pressure. In this study, we compared the effects of increased intraabdominal pressure during CO_2 pneumoperitoneum on cardiac function in patients undergoing laparoscopic and open GBP. To eliminate confounding variables, similar amounts of intraoperative fluid (preload) were administered, ventilatory adjustments were performed during pneumoperitoneum to prevent systemic hypercarbia, and similar intraoperative operative positioning was used for both groups.

The present study demonstrated that cardiac output (CO) decreased slightly by 5.7% at 1 h after abdominal insufflation during laparoscopic GBP. This reduction in CO, however, was not statistically significant when compared with baseline values. The CO recovered and increased significantly by 5.3% at 2.5 h after insufflation. During open GBP, CO was increased by 25.3% at 0.5 h and remained elevated throughout the operation. Desufflation after laparoscopic GBP resulted in a marked increase of CO by 42.8%, which was similar to the increase (52.1%) seen after open GBP. Our study confirmed that with proper attention to intraoperative acid–base status and maintenance of an

euvolemic state, prolonged CO_2 pneumoperitoneum does not significantly depress cardiac function in morbidly obese patients.

Clinical studies to evaluate the effects of CO_2 pneumoperitoneum on cardiac function using Swan-Ganz or transesophageal echocardiography (TEE) have recorded varied results. Several investigators have demonstrated a reduction in CO during pneumoperitoneum [1, 7, 12, 15], whereas others have reported no change [2, 4, 6, 11, 13]. McLaughin et al. [12] reported a 29.5% decrease in cardiac index in patients undergoing laparoscopic cholecystectomy. Ninomiya et al. [15] reported a reduction of CO during laparoscopic cholecystectomy when compared with cholecystectomy performed using an abdominal wall-lifting device. Dexter et al. [1] reported a 25% reduction in CO after high-pressure (15 mmHg) insufflation compared to low-pressure (7 mmHg) insufflation. Joris et al. [7] also found a 33% reduction in cardiac index immediately after pneumoperitoneum, which also resolved after desufflation. Conversely, Kraut et al. [11] and Dorsay et al. [2], using TEE, reported no change in CO in patients receiving 15 mmHg of insufflation during laparoscopic cholecystectomy. Inabnet et al. [6] also found no difference in cardiac index between patients undergoing laparoscopic and open adrenalectomy. Hirvonen et al. [4], using Swan-Ganz catheterization, reported a transient but nonstatistically significant decrease in cardiac index during laparoscopic cholecystectomy, which resolved on desufflation. All of these studies, however, were performed during short laparoscopic procedures such as cholecystectomy and adrenalectomy. By contrast, our study evaluated the cardiovascular response to prolonged pneumoperitoneum in patients undergoing laparoscopic and open GBP.

Several mechanisms have been proposed to explain the reduction in cardiac function observed after pneumoperitoneum. These mechanisms include hypercarbia, the head-up tilt position, hypovolemia, and increased intraabdominal pressure. Using an animal model, Ho et al. [5] attributed the observed cardiovascular depression to systemic acidosis. Absorption of CO_2 can lead to an increased $PaCO_2$, which induces myocardial depression, increased catecholamine, and vasodilatation. However, Shuto et al. [16] showed that helium insufflation also reduced CO, which suggested that increased intraabdominal pressure might be, in part, the cause for reduced CO. In the present study, $PaCO_2$ levels at 3 h after surgical incision were significantly higher for laparoscopic GBP than for open GBP (42.1 ± 4.8 vs 36.6 ± 4.4 mmHg, $p = 0.001$). However, it is unlikely that this level of $PaCO_2$ would be a major factor affecting cardiac function. Reverse Trendelenburg position was shown by Joris et al. [7] to reduce cardiac index by 18% when compared to the supine position in healthy adults. To eliminate positioning as a confounding variable, we standardized the two groups by placing all patients in the reverse Trendelenburg position during surgical dissection of the gastric pouch. Despite the position, CO increased during open GBP, which demonstrates that reverse Trendelenburg positioning is not a major factor affecting intraoperative CO.

The combination of decreased preload and increased intraabdominal pressure may also account for the observed reductions of CO during pneumoperitoneum. "Preload" is defined as the force acting upon the cardiac muscle before contraction. The Frank-Starling law states that there is a relationship between the initial length of cardiac muscle fiber and the force of cardiac contraction. A low preload combined with an increased intraabdominal pressure may impede venous return and depress cardiac function. In our study, both the laparoscopic and open GBP groups received a similar amount of intraoperative fluid, but the operative time was shorter in the open group. Taking operative time into account, open GBP group received slightly more intraoperative fluid (1.65 L/h for the open group vs 1.44 L/h for the laparoscopic group) to compensate for the higher intraoperative blood loss. An euvolemic state was maintained in both groups throughout the operation, as indicated by the stable CVP and PAWP measurements. During laparoscopic GBP, there was a transient increase in MPAP and CVP after insufflation, whereas PAWP remained stable in the range of 17–19 mmHg throughout the operation. This elevation of filling pressures during laparoscopic GBP might have been exacerbated by the elevation of intrathoracic pressure from the increased intraabdominal pressure. During open GBP, there was a transient decrease in MPAP, CVP, and PAWP, but the PAWP was still in the range of 14–18 mmHg. The PAWP levels in both Hirvonen et al.'s [4] and Joris et al.'s [7] studies, compared with the PAWP levels in our study, were low (3–9 mmHg) before insufflation; thus, the low preload may have contributed to the decrease in cardiac function observed immediately after insufflation.

Changes in CO are also related to an increase in heart rate. In the present study, HR increased immediately after surgical incision by 3–11% in the open GBP group to reach a maximal rate of 82 ± 13 beats/min. During laparoscopic GBP, HR remained unchanged from baseline value at 73 ± 15 beats/min until 2 h after insufflation, when mean HR increased to 79 ± 12 beats/min. The faster HR during open GBP may represent a higher catecholamine release in response to surgical stimulation than during laparoscopic GBP and may have contributed to the higher CO seen in these patients.

Afterload, which is the arterial resistance against which the left ventricle must pump, is another determinant of cardiac function. Afterload is reflected in the SVR. An increase in SVR can result in a decrease in SV and thus a decrease in CO. Our results showed a transient increase in SVR immediately at 0.5 h after insufflation compared to baseline value (975 ± 425 vs 727 ± 179 dynes • s • cm^{-5}, respectively, $p = 0.005$). The SVR returned to within baseline value by 1.5 h. This transient increase in SVR is probably not related to the head-up tilt position, because both the laparoscopic and open GBP patients were in a head-up tilt position during the early stages of the operation. It most likely reflects the increased intraabdominal pressure or systemic absorption of CO_2. The increase in SVR may explain the transient but nonsignificant decrease in CO during laparoscopic GBP measured at 0.5 h and 1 h. Kashtan et al. [9] also found that an increased intraabdominal pressure led to elevation of arterial and venous resistance and therefore SVR. Shuto et al. [16] reported an increase in total peripheral resistance with increasing intraabdominal pressure in a swine model. They found that changes in total peripheral resistance were similar in animals receiving helium or CO_2 insufflation, suggesting that the mechanism of increasing total peripheral resistance is unrelated to CO_2 absorption.

In conclusion, our results show that prolonged CO_2 pneumoperitoneum at 15 mmHg does not produce major hemodynamic changes in morbidly obese patients. Laparoscopic GBP was associated with a transient increase of CVP, MPAP, and SVR, but CO remained unchanged as long as a low systemic level of CO_2 and adequate intravascular volume status was maintained. When comparing CO during open GBP with CO during laparoscopic GBP, we observed a lower CO during laparoscopic GBP at 0.5 h and 1 h after insufflation. The mechanism for this change is likely related to the immediate effect of elevated intraabdominal pressure that increased SVR. The systemic effect of increased intraabdominal pressure during laparoscopic GBP was compensated for after 1 h as SVR returned to baseline values by 1.5 h. This randomized trial with a control group confirmed that, with proper attention to adequate intravascular volume resuscitation and a normal acid-base status, one can avoid the potential depression of cardiac function during prolonged laparoscopic operations. We must emphasize that the results of this study should be viewed in the context of the trial. Our patient population was morbidly obese but had no history of myocardial infarction or coronary artery disease. We have no data regarding the effects of pneumoperitoneum on patients with compromised cardiac function.

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