ANAESTHESIA FOR LAPAROSCOPIC BARIATRIC SURGERY – OUR EXPERIENCE

Dr. Jayashree Sood[1] Dr. Lakshmi Jayaraman[2] Dr. Nitin Sethi[3]

SUMMARY

Bariatric surgery is the most effective method for treating patients with morbid obesity. These patients are a unique challenge for the anaesthesiologist because of the various anatomic and physiological changes in the body and associated co-morbid conditions. The present study describes a series of 123 patients with an average body mass index of 48.4 kg/m^2 who underwent laparoscopic bariatric surgery in the form of gastric bypass, sleeve gastrectomy and gastric banding at our institution.

Keywords : Bariatric surgery, Body mass index, Morbidly obese.

Introduction

Morbid obesity is one of the most prevalent medical disease states in the world. Surgery performed for the treatment of morbid obesity, commonly referred to as bariatric surgery, has allowed a large population the opportunity to receive treatment for their disease. In the 21st century, Roux-en-Y gastric bypass, and sleeve gastrectomy are considered the gold standard in bariatric surgery, it produces massive weight reduction (70% or greater) with maintenance of 50% or more excess weight loss beyond 10 years. The perioperative care of clinically severe obese patients presents numerous unique challenges. As almost all the organs of the body are affected, these patients have unique issues with regards to airway, cardiovascular, pulmonary gastrointestinal, endocrine and psychiatric complications. To have excellent outcome a multidisciplinary approach, including primary care physician, anaesthesiologist, surgeon, nursing staff, dietician and social worker is necessary.

We present a series of 123 patients who underwent laparoscopic bariatric surgery at our institution. The laparoscopic bariatric procedures performed included Roux-en-Y-gastric bypass, sleeve gastrectomy and gastric banding.

Laparoscopic adjustable gastric banding – In this procedure a band is placed around the stomach just below the esophago-gastric junction and tightened below an intragastric calibration balloon filled to 15 ml with saline.

1. M.D., FFARCS, PGDHHM, Sr. Consultant & Chairperson
2. D.A., D.N.B., MNAMS, Asso. Consultant
3. D.N.B., Sr. Registrar
Dept. of Anaesthesiology, Pain & Perioperative Medicine
Sir Ganga Ram Hospital, New Delhi - 110 060, INDIA.
Correspond to :
Dr. Jayashree Sood
E-mail : jayashreesood@hotmail.com

Roux-en-Y-gastric bypass – This procedure involves anastomosing the proximal gastric pouch to a segment of the proximal jejunum bypassing most of the stomach and entire duodenum.

Sleeve gastrectomy – In this procedure a polypropylene band is placed around the lesser curvature of the stomach and four to six vertical rows of staples are aimed near the gastroesophageal junction; this creates a small pouch along the lesser curvature and a narrow passage into the larger remainder of the stomach.

Materials and Methods

123 patients who underwent laparoscopic bariatric procedures between December 2004 and May 2007 were included in our study. The age varied between 32-58 years, were of either sex and belonged to ASA II –IV.

In the preanaesthetic check up body mass index (BMI) was calculated and the patients were evaluated for hypertension, coronary artery disease, diabetes mellitus, sleep apnea syndrome or any other coexisting medical problem, which was stabilized before surgery. A detailed airway assesment was also done. A suitable site for intravenous access was noted.

Preoperative investigations included haemoglobin, haematocrit, blood sugar, blood urea nitrogen, serum creatinine, serum electrolytes, thyroid function tests, liver function tests, arterial blood gases, pulmonary function tests, chest radiography, electrocardiogram, echocardiography (ECHO) stress echo and polysomnography (in patients with obstructive sleep apnea).

Patients were premedicated with tab. metoclopramide 10 mg, tab. ranitidine 150 mg and tab. alprazolam 0.25 mg night before and on morning of surgery. Sedative premedication was avoided in patients with obstructive sleep apnea. DVT prophylaxis was given to all patients with inj. enoxaparin 40 mg s/c on morning of surgery.

On the day of the surgery, the patients were wheeled into the theatre on a special trolley accepting weight > 220Kg. Intravenous access was obtained with a 20 gauge intravenous cannula. Before induction of anaesthesia a central venous and radial artery cannulation was done under local anesthesia for intraoperative continuous monitoring. Other monitors attached intraoperatively included pulse oximeter, non-invasive blood pressure (using large size BP cuff which encircled ¾ of the upper arm), $EtCO_2$, electrocardiogram (ECG), bispectral index (BIS) monitor, peripheral neuromuscular monitor (TOF guard) and temperature probe. Sequential pneumatic compression devices were applied on the calves of the patients along with graduated compression stockings (Fig. 1).

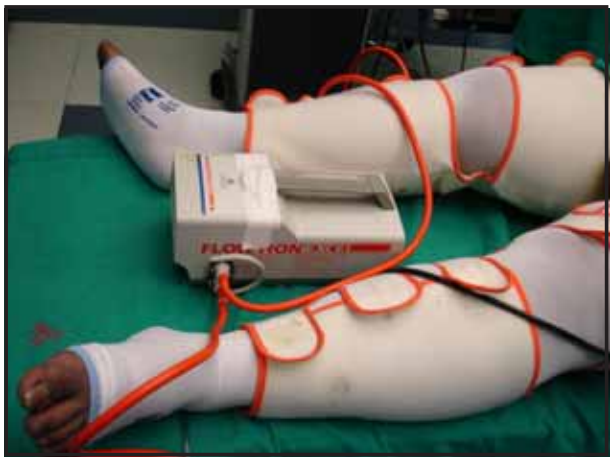

Fig. 1

In patients with normal clinical evaluation preoxygenation with 100% O_2 was done for 5 minutes. Inj. atracurium besylate 5 mg was given for precurarization and anaesthesia induced with sleep dose of propofol. Fentanyl citrate (1-2 mg/kg) was given for intraoperative analgesia followed by succinylcholine 2mg/kg. Trachea was intubated with appropriate sized endotracheal tube. Endotracheal intubation was done using an standard intubation pillow in conjuction with an elevation pillow (ramp position) (Fig. 2). This was done to increase the sternomental distance so as to facilitate tracheal intubation. After intubation a esophageal calibration tube was inserted to decompress the stomach and the urinary bladder catheterized to empty the bladder (Fig. 3).

Anaesthesia was maintained with O_2 + N_2O and propofol infusion titrated to maintain a BIS value of 40-60. Atracurium infusion was started at 10ìg/kg/min and was titrated to maintain a single response to train of four stimuli throughout the duration of surgery. Ventilatory settings were adjusted to maintain minute ventilation to keep $EtCO_2$ between 35-40 mmHg and SpO_2 between 95-100%. Positive

Fig. 2

end-expiratory pressure (PEEP) of 5-7cm H_2O was given to all patients. All pressure points were adequately padded. Carboperitoneum was initiated and intraabdominal pressure maintained between 15 to 20 mmHg. The surgery was carried out in modified Lloyd Davis position (steep reverse Trendelenberg position with legs spread apart).

Fig. 3

At the end of the procedure atracurium and propofol infusions were discontinued. When the neuromuscular monitor showed 50% response to train-of-four stimuli, the residual neuromuscular block was reversed with inj. neostigmine methylsulphate0.04 mg/kg and inj. glycopyrrolate sulphate 0.01 mg/kg.

Tracheal extubation was performed when the patients showed adequate clinical signs of reversal of neuromuscular block and train-of-four response had returned to 90% of control. Patients were shifted from the OT table to the trolley was done with the help of patslide (Fig. 4).

Fig. 4

The patients were shifted postoperatively into the high dependency unit or intensive care unit depending on the Montefiore Obesity Surgery Score (Table 1)

Table 1 : Montefiore Obesity Surgery Score

Class	Description	Disposition
I	< 40 years	If stable in PACU
	No major respiratory problem	4hrs postoperatively, can go to ward
II	40-50 years H/O asthma/snoring	Oximetry 24 hrs HDU observation
III	> 50 years OSA, asthma, DVT	Overnight HDU ICU if there are complications

Postoperative pain relief was provided with local anaesthetic wound infiltration of port sites with 0.125% bupivacaine hydrochloride and inj. diclofenac sodium 75mg intramuscularly eight hourly. On the first postoperative day, oral gastrograffin study was done to assess the leak. If no leak was present, then oral nimuselide mouth dissolving tablets were started three times a day along with H_2 receptor blockers.

The patients were made ambulatory on 1st postoperative day and were discharged from the hospital between 3rd to 5th postoperative day.

Results

The study included 123 patients, 39 women (32%) and 84 men (68%) whose average age was 42+10 years (range 32-58 years). The average weight was 146 + 33 kg (95-220 kg), height 160 + 10 cm and BMI 48.4 +10 kg/m^2 (range 35-76 kg/m^2). 25 patients (20 %) had a BMI less than 40 kg/m^2, 34 patients (28%) had BMI in the range 40-45 kg/m^2 and 64 patients (52%) had a BMI greater than 45 kg/m^2 (Table 2). All the patients had some concomitant disease, systemic arterial hypertension in 72%, coronary artery disease in 52%, diabetes mellitus in 52%, and obesity-hypoventilation syndrome in 56% and obstructive sleep apnea syndrome in 64% (Table 4). 39 patients (32%) were classified ASA grade II, 58 patients (48%) as ASA grade III and 26 patients (20%) as ASA grade IV.

Table - 2 : Demographic profile

	n = 123
Age (yrs)	42 ± 10
Sex (M/F)	84:39
Weight (Kg)	146 ± 33
Height (Cms)	160 ± 10
BMI (Kg/m^2)	48.4 ± 10

Table - 3 : Type of surgery

Roux-en-Y-gastric bypass	56
Sleeve gastrectomy	41
Laparoscopic adjustable gastric banding	26

Table - 4 : Associated co-morbidity

Disease	No. (%)
Systemic arterial hypertension	88 (72%)
Coronary artery disease	64 (52%)
Diabetes Mellitus	64 (52%)
Low back ache	44 (36%)
Rheumatoid arthritis	49 (40%)
Gastroesophageal reflux	39 (32%)
Obesity Hypoventilation syndrome	69 (56%)
Obstructive Sleep apnea	79 (64%)
Hypothyroidism	74 (60%)

The pre-operative laboratory tests are shown in Table 5.

79 patients (64%) had normal chest radiographs. Mild cardiomegaly was present in 44 patients (36%) and 14 (11%) of them also had radiological signs of pulmonary hypertension. 29 patients (24%) had one of the following ECG abnormalities: left ventricular enlargement, subepicardial ischemia of the left ventricular free wall,

bundle branch block, right ventricular overload, non-specific repolarization disorders and ventricular bigeminy. By echocardiogram and ECG evaluation, left ventricular enlargement was documented in 26 patients (20%). 74 patients (60%) had normal spirometry, 29 patients (24%) had obstructive airflow and 19 patients (16%) had restrictive pattern of airflow.

Table - 5 : Preoperative laboratory tests

Test	Mean + SD	Range
Haemoglobin (g/dl)	15.2 + 2.4	10.5 –23.0
Hematocrit (%)	46.2+ 8.8	32-74
Glucose (mg/dl)	143 + 63	76-419
Creatinine (mg/dl)	0.88 + 0.21	0.2-1.2
Blood urea nitrogen (mg/dl)	13.7+ 2.9	8-20
Total bilirubin (mg/dl)	0.85 + 0.4	0.2-2.0
Direct bilirubin (mg/dl)	0.23 + 0.24	0 –1
Alanine transaminase (IU/dl)	31+ 16	10-66
AST (IU/dl)	29 + 13	11-82
Akaline phospatase (IU/dl)	96 + 24	33-156

In the airway assessment, 44 patients (36%) were classified as Mallampati I, 19 (16%) as Mallampati II, 34 (28%) as Mallampati III and 26 (20%) as Mallampati IV.

Table - 6 : Comparison of BMI and ASA classification.

	Percentage over ideal body weight	BMI	ASA class
Ideal Body Weight	0	20-25	I
Minimally Obese	1-19	26-29	II
Morbid Obese	20-100	30-35	III
Super Morbid Obese	200	> 35	IV

In 88% of the patients, endotracheal intubation was performed after a rapid sequence induction, while 12% were intubated with a fibreoptic bronchoscope after local anaesthesia of airway. Tracheal intubation was successful in all patients with no incidence of gastric aspiration or any other complication during intubation.

In all patients the trachea was extubated in the operating room at the end of the surgery. Only 26 (20%) patients with history of OSA required CPAP in the post-operative period (Fig. 5).

Fig. 5

No post-operative complications were noted in any patient. They were made ambulatory on the first postoperative day and discharged from the hospital between the third and fifth postoperative day. However 2 patients developed anastomotic leak 5 days postoperatively and were managed conservatively.

Discussion

We report our experience with 123 patients who underwent laparoscopic bariatric surgery at our institution. Morbid obesity exists when the BMI is more than 35kg/m^2 and super morbid obesity when BMI is more than 55kg/m^2. The American Society of Anaesthesiologists (ASA) risk classification is also influenced by a person's weight (Table 6). Studies have reported upto 12-fold increase in mortality rate in association with morbid obesity along with an exceptionally high incidence of co-morbodities.[1] The primary goal of bariatric surgery is improvement of obesity related co-morbidities. Surgery is a viable option for weight loss in patients with:

- BMI equal to or greater than 40 kg/m^2 who have failed serious attempts at non-surgical treatment.
- BMI equal to or greater than 35 kg/m^2 who have failed non-operative treatment and have serious medical co-morbidities.[2]

In our case series average BMI of patients was 48.4 kg/m^2 and all our patients had some associated co-morbidity.

Given the frequency of anatomic and physiological alterations associated with morbid obesity and existence of concomitant diseases, the goal of preoperative evaluation is to identify risk factors that may modify perioperative course.[3,4,] The evaluation begins with revision of the

complete health history, a detailed physical examination, and baseline laboratory screening that includes blood cytology, glucose, routine urine test, serum electrolytes, renal and liver function tests, blood gases, chest radiography and ECG.

One aspect of paramount importance is evaluation of airway, whose management is critical.[5,6] Endotracheal intubation can be a daunting experience in the morbidly obese patient. Neither obesity nor BMI predicts problems with tracheal intubation.[7] A high Mallampati score (> 3) and large neck circumference may increase potential for difficult laryngoscopy and intubation.[8] It is worthwhile to emphasize the need to have appropriate equipment to manage a difficult airway: special laryngoscopes, laryngeal masks, fiberoptic bronchoscope and even instruments for a surgical access.[9]

Evaluation is initiated with anaesthetic history, physical examination and appropriate preoperative investigations. Physical examination identifies abnormalities inherent to morbid obesity, which could interfere with maintaining airway patency, mask-ventilation and endotracheal intubation. Mallampati assessment evaluates mouth opening and the relation between the oral cavity and soft tissues. Frequently, because of cervical fat, tongue size, amount of palate folding, and laryngeal and supralaryngeal tissue, the mouth opening and cavity-soft tissue relation is altered. In our series, 36% of the patients were assessed as Mallampati I, 16% as Mallampati II, 28% patients as Mallampati III and 20% patients as Mallampati IV. Frequently, also, cervical mobility and ability of the patient to adopt a "sniffing the morning air" position is altered by fat accumulated at the cervical and thoracic levels. Moreover, excessively large breasts may render difficult movement of the laryngoscope. Anatomic difficulties and physiological changes, such as an increase in oxygen consumption and CO_2 production in addition to lower pulmonary reserve of oxygen cause a rapid fall in oxygen arterial saturation. Due to the aforementioned reasons, it is important to have a strategy for securing the airway.

Obesity produces a variety of cardiac, structural and haemodynamic alterations. It is pathogenically related to the development of atherosclerotic plaques and thrombosis, hypertension, cardiomyopathy and arrhythmias.[10] Hyperkinetic circulation, myocardial hypertrophy, decreased compliance and increased systemic arterial pressure ultimately results in diastolic dysfunction.[11] As the capacity of the heart to hypertrophy is limited systolic dysfunction ensues and the resultant clinical syndrome of obesity cardiomyopathy predisposes to arrhythmias and sudden death. Plasma renin and aldosterone levels are increased in obese subjects. For this reason, a cardiovascular evaluation is mandatory. Pharmacological treatment of concomitant diseases must be optimized before surgery. For those patients who have other risk factors, clinical data or ECG alterations compatible with ischemia must be assessed with specialized studies such as stress echocardiography, scintigraphy studies and thallium dipyridamole or even coronary arteriography.[12]

Obstructive sleep apnea (OSA) is frequently found in morbidly obese patients[13,14] and in our series 64% presented this complication. OSA may contribute to pulmonary arterial hypertension. It must be suspected in all cases and if there are suggestive findings, the diagnosis must be confirmed. Likewise, and in direct relation to the degree of overweight, the obesity-hypoventilation syndrome (OHV) is quite prevalent (56% in our series), and it must always be sought intentionally. When OSA diagnosis is confirmed nasal continuous positive airway pressure (PAP) is initiated, which has significantly reduced the need for tracheostomy.[14]

Drug pharmacokinetics is altered in obese patients and it depends on changes in volume of distribution, protein binding properties, increased renal clearance and changes in liver clearance.[15] It is recommended that drugs with a short half-life be used to guarantee spontaneous ventilation once the medication is stopped. It is also essential to monitor the degree of neuromuscular block to ensure that there is no residual effect on completion of surgery.

Intraoperative monitoring must follow the basic ASA guidelines.[16] We stress the importance of closely monitoring respiratory function with emphasis on airway pressure, tidal volume capnography and pulse oximetry. All obese patients should have their ventilation controlled with a minute volume of 70-80 ml/kg. This large volume coupled with low chest wall compliance and elevation of the diaphragm means that high peak inspiratory pressures are often necessary. Ventilation with large tidal volumes aims at moving tidal ventilation above the closing volume and consequently increasing arterial oxygen tension. Application of moderate positive end expiratory pressure can improve oxygenation.[17] Adverse postoperative respiratory events are twice as likely to occur in obese patients compared with non-obese patients. These patients are at high risk for aspiration pneumonia. Atelectasis is also common in postoperative period. This is the result of pneumoperitoneum in the laparoscopic approach. Atelectasis can be associated with fever and tachycardia during the initial 24 hours. Treatment options include incentive spirometry, continuous positive airway pressure and pulmonary toilet.[18]

There were no postoperative complications present in our series of 123 patients. However 2 patients had

anastomotic leak 5 days postoperatively and were rept on conservative management.

In conclusion, the treatment for patients with morbid obesity who undergo surgery represent a great challenge. It requires a multidisciplinary approach with a team of physicians, surgeons, anaesthesiologists, nursing staff and dietician with depth of knowledge about the physiopathological implications of morbid obesity and with experience in their management.

References

1. *Dresnick EJ, Bale GS, Seltzer F et al.* Excessive mortalty and cause of death in morbidly obese men. *JAMA* 1980; 243: 433-35.
2. National Institute of Health Consensus Development Conference Panel. Gastrointestinal surgery for severe obesity. Ann Intern Med 1991; 115: 956-61.
3. *Shenkamen Z, Shir Y, Brodsky JB.* Perioperative management of the obese patient. Br J Anaesth 1993; 70: 349-361.
4. *Luce JM.* Respiratory complications of obesity. Chest 1980; 78: 626-630.
5. *Mallampti SR, Gott SP, Gugino CD, et al.* A clinical sign to predict difficult tracheal intubation: A prospective study. Can Anaesth Soc J 1985; 32: 429-433.
6. *Jacobsen J, Jenson E, Waldov T, Poulsen DT.* Postoperative evaluation of intubation conditions in patients scheduled for elective surgery. Acta Anaesthesiol Scand. 1996; 40: 425-434
7. *Brodsky JB.* Morbid obesity and tracheal intubation. Anesth Analg 2002; 94: 732-6.
8. *Levitan R, Ochroch EA.* Airway management and direct laryngoscopy- a review and update. *Crit Care Clinic* 2000; 16: 373-88.
9. *Reed AP.* The unanticipated difficult airway. Anaesthesiol Clin North Am 1996; 14: 443-452.
10. *De Vitiss O, Fuzio S, Peleto M, et al.* Obesity and cardiac function. Circulation 1981; 64: 477-80.
11. *Alport MA, Hashmi MW.* Obesity and the heart. Am J Med Sci 1993; 306: 117-23.
12. *Buckley FP.* Anaesthetic management of the obese patients in Anaesthesia (2nd edition), WS Nimmo, PJ Radbotham, G Smith Editors: Oxford, Blackwell Scentific, 1994, pp. 1148-1160.
13. *Shepard JW.* Cardiopulmonary consequences of obstructive sleep apnea. Mayo Clin Proc 1990; 65: 1250-1257.
14. *Charuzi I, Fraser P, Pesser J et al.* Sleep apnea syndrome in the morbidly obese undergoing bariatric surgery. Gastroenterol Clin North Am. 1987; 16: 517-523.
15. *Abernethy DR, Greenblatt DJ.* Drug disposition in obese humans: An update. Clin Pharcokinet. 1986; 11: 199-213.
16. American Society of Anesthologists 1998 Directory of Members, Washington D.C., American Society of Anaesthologists, 1998; pp. 438-439.
17. *Fox GS.* Anaesthesia for intestinal short-circuiting in the morbidly obese with reference to pathophysiology of gross obesity. Can Anaesth Soc *J* 1975; 22: 307-315.
18. *Marik P, Varon J.* The obese patient in the ICU. Chest 1998; 113: 492-8.