Management of Obesity: The Surgical Perspective

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ABSTRACT

Background: Obesity which is one of the most prevalent diseases worldwide continues to increase with its associated complications. Bariatric surgeries have been described as the preferred method of treatment for morbid obesity. The objective of this review is to give an overview of bariatric surgical procedures and the possible outcomes.

Method: Literature review was conducted using Goggle Search Engine and Highwire Press. Selected papers were taken and books from the author's collection were used for further reference.

Results: Bariatric surgery is the most effective treatment for morbid obesity producing long lasting weight loss and remission of co-morbidities. Restrictive procedures are more commonly performed because they are simpler and achieve good weight loss in addition to the avoidance of metabolic complications associated with malabsorptive procedures.

Conclusion: Specific criteria must be met and the patient must be motivated and fully informed before performance of bariatric surgery for obesity preferably with restrictive procedures.

Key Words: Obesity; bariatric surgery; restrictive; malabsorptive

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INTRODUCTION

Obesity is a state of excess adipose tissue mass resulting in Body Mass Index (BMI) of 30kg/m^2 or greater.

BMI is calculated by dividing the weight (in kilograms) by the square of height (in meters). BMI between 25 and 29.9kg/m^2 is termed overweight. Morbid obesity is BMI of 40kg/m^2 or greater[1].

There are other indices used to identify obesity such as the waist-hip ratio which is the ratio of waist circumference (measured at midpoint between the lower margin of the last palpable rib and the top of ileal crest) to hip circumference (measured around the widest portion of the buttocks).

The ideal body weight which is 50kg + 2.3kg for every inch over 5ft for men and 45.5kg + 2.3kg for every inch over 5 ft for women can also be used as an index of obesity.

Obesity is at present a major health problem and one of the most prevalent diseases globally which imposes devastating health and financial burden on the victims and society.

Despite the increasing awareness of this burden, obesity with its co-morbidities continues to increase at an alarming rate[2]. Many reports have emphasized the alarming increase in the prevalence of obesity and overweight in the United States of America[3-5]. A study estimates the combined prevalence of obesity and overweight to be over 64% of the United States (US) adult population with 4.7% as morbid obesity[5]. In the US, obesity and its complications result in about 300,000 deaths annually[6].

As a result of the increase in incidence, treatment options for obesity have become more numerous including non medical options, with increasing rates of bariatric surgery as shown in California where the number of bariatric surgeries rose from 1134 in 1996 to 6304 in 2000[7].

The prevalence rates of obesity and its co morbidities like hypertension, diabetes and dyslipidaemia in developing countries are relatively low but are changing rapidly with urban and rural variations[8]. In Nigeria, the prevalence rate reported from a study in Okrika in Rivers State was 16.3%[9]. Paradoxically, the access to bariatric surgeries in developing countries like Nigeria is limited and rare.

The objective of this review is to give an overview of bariatric surgical procedures, evaluate the possible outcomes, recommend preferred methods and create awareness among surgeons on the expected increase in demand for bariatric surgeries by patient s in Nigeria.

Obesity: Pathogenesis and complications

Obesity is the result of multifactorial changes involving both environment and genetic factors. Accumulation of fat results when energy consumption exceeds energy expenditure over a long period of time and the discrepancy becomes too large to be detected by the hypothalamic regulation of the Basal Metabolic Rate[10].

Ghrelin is the first circulating hunger hormone. It is a hormone produced mainly by the P/D1 cells lining the fundus of the stomach and epsilon cells of the pancreas that stimulate hunger[11]. The levels which increase before meals and decrease after meals are lower in the plasma of obese than leaner individuals. In some bariatric procedures, the level of ghrelin is reduced in patients causing satiation before it would normally occur. Ghrelin increases food intake and fat mass[12] by an action exerted on the hypothalamus. It activates cells in the arcuate nucleus[13] that include the orexigenic neuropeptide Y(NPY) neurons[14]. Ghrelin responsiveness of these neurons is both leptin and insulin sensitive. It also activates the mesolimbic cholinergic-dopaminergic reward link, a circuit that communicates the hedonic and reinforcing aspects of

natural rewards, such as food[15] leading to increase fat mass. Genetic factors have led to the discovery of ob gene which is expressed solely in both white and brown adipose tissue. The gene is found on chromosome 7 and produces leptin[16]. Leptin acts as a feedback mechanism between the adipose tissue and the brain, acting as lipostat controlling fat stores by regulating hunger and satiety[16]. High leptin levels decrease food intake and increase energy expenditure. Mutation in either leptin or the leptin receptor has led to the development of morbid early onset diabetes in several families[10]. Rarely, mutation in some other genes cause obesity in human's e.g mutation in tub gene which encodes a hypothalamic peptide of unknown function, causes late onset diabetes[10]. A number of complex human syndromes have also been associated with obesity such as Prader-Willi syndrome, Ahlstrom syndrome and Cohen syndrome.

Morbid obesity is very likely to have adverse effect on an individual's health and is defined as BMI of 40kg/m^2 or greater or 35kg/m^2 or greater in association with weight related co-morbidities which may include hypertension, type 2 diabetes, coronary heart disease, dyslipidaemias, non-alcoholic hepatic steatosis, obstructive sleep apnoea, osteoarthritis, reduced fertility and a greater susceptibility to a range of cancers[17]. Reduced life expectancy has been documented in obese patients irrespective of ethnicity, age or sex[18].

Treatment of Obesity:

The treatment options include diet and exercise programme, the use of drugs and bariatric surgery.

Diet and exercise programme: it is the first step in the process of losing weight. Nevertheless, in patients with morbid obesity, their value is limited. This is because their success rates are not consistent and their rates of weight regain are high[19].

Use of drugs: this is relatively common in the management of obesity. Orlistat and sibutramine are the only drugs licensed for this purpose[20]. Orlistat is a saturated derivative of lipstatin, a potent natural inhibitor of pancreatic lipase. It inhibits the conversion of dietary fat into its absorbable form, thereby, preventing its absorption. Sibutramine creates a feeling of satiety by centrally inhibiting serotonin and noradrenalin reuptake. In 2005, a meta-analysis of drug treatment for obesity showed a mean weight loss of 2.9kg in one year for orlistat and 4.5kg for sibutramine and this was insufficient to make any clinical difference[21]. It was observed that when therapy is stopped, weight is almost invariably regained.

Bariatric surgery: this is the most effective treatment for morbid obesity producing long lasting weight loss and improvement or remission of co morbid conditions[22]. In a randomized controlled trial comparing a group of patients undergoing pharmacological treatment with a matched group of patients undergoing obesity surgery, it was observed after 8 years of follow-up that those who had bariatric surgery lost an average of 20kg while those treated medically did not lose any weight compared with baseline[23].

In 2006, the National Institute for Health and Clinical Excellence (NICE) produced a set of guidelines for selection of patients for obesity surgery[24]. These include:

a) The patient must have a BMI ≥40Kkg/m^2 or ≥35kg/m^2 associated with serious obesity related co morbidity (type 2 diabetes, hypertension, obstructive sleep apnoea).
b) All non-surgical methods must have been tried and failed to produce clinically significant weight loss over a 6 month period.
c) The patient will receive intensive follow-up in a specialist service.
d) The patient is considered fit for anaesthesia.
e) The patient commits to the need for long-term follow-up.

These guidelines also recognized that in patients with a BMI>50kg/m^2, surgery can be offered without a trial medication[18]. The goals of surgery are to achieve and maintain significant weight loss and to reverse or prevent many of the obesity induced co morbidities. Bariatric surgery was first performed in the early 1960s and its use has increased dramatically, particularly in recent years. The surgical procedures are broadly divided into 2 groups: the Restrictive and Malabsorptive procedures[25].

RESTRICTIVE PROCEDURES

These procedures decrease the storage capacity of the stomach. A small stomach pouch is associated with prompt filling by a small amount of food, early satiety, decrease meal size and calorie intake, ultimately resulting in weight loss. Restrictive procedures are simpler to perform and they achieve good weight loss. The common restrictive procedures

Figure 1: Roux-en-y Gastric Bypass

Diagram is adapted from Benjamin et al[25]

are Roux-en-y Gastric Bypass, Gastric Banding, Vertical Banded Gastroplasty and Sleeve Gastrectomy. (Figure 1 — 4)

Roux-en-y Gastric Bypass

It is a procedure in which a small upper stomach pouch is fashioned and divided from the remainder of the stomach. Additionally, there is intestinal reconstruction which bypasses a portion of the intestine thereby, creating some degree of malabsorption and may result in dumping syndrome. This syndrome occurs following ingestion of high carbohydrate meal and may result in diaphoresis, nausea, palpitations, diarrhoea, abdominal pain or light headedness[26]. Recently, laparoscopic techniques have been applied to accomplish the same procedure in an effort to reduce post operative morbidities and improve the quality of life[27]. Bypassing the fundus of the stomach which is the major production site of ghrelin results in significant decrease in its serum level and in turn the patient's apetite[28].

Early complication rate of 10% and mortality rate of 1% have been reported[29]. Complications may include bleeding, incisional or internal hernias, anastomotic leaks, strictures, thromboembolic events, marginal ulcerations, gall stone formation, surgical site infection, vitamin and protein malnutrition. These complications become reduced as the learning curve increases following laparoscopic surgeries[30]. Lifelong follow-up of these patients is required in order to avoid vitamin and mineral deficiencies.

Some studies have shown that this procedure improves or resolves glucose intolerance and type 2 diabetes almost immediately in the majority of patients even before weight loss is observed[31-32]. Other co morbid conditions such as

Figure 2: Gastric Banding

Diagram is adapted from Benjamin et al[25]

hypertension(52-91.5%), sleep apnoea (74-97.8%) and hypercholesterolemia(63-97%), also have been noted to resolve[33].

Gastric Banding

This is the least invasive among the frequently performed procedures. It consists of a silicon elastomer with an adjustable inner balloon that effectively allows for control of the stomal aperture. The band encircles the proximal stomach to create a very small(15-20mls) pouch that effectively restricts the amount of food ingested. Adjustments are later performed at the bed side by a needle accessing of the subcutaneous port which is sutured to the abdominal wall fascia.

The laparoscopic approach is standard with a very low conversion rate to the open approach(0.3-1%)[34]. It has the lowest complication rate of 0-0.7% among all the bariatric procedures[35]. In 2.8% of patients, erosion of the band into the gastric wall may occur[35] while in 2-14.2% of patients, band prolapse or slipping of the band from its intended site may occur[36]. Revision of the band will be required in 1-13% of patients[36]. Weight loss has been found to be 34.5-58% in 12

Figure 3: Vertical Banded Gastroplasty

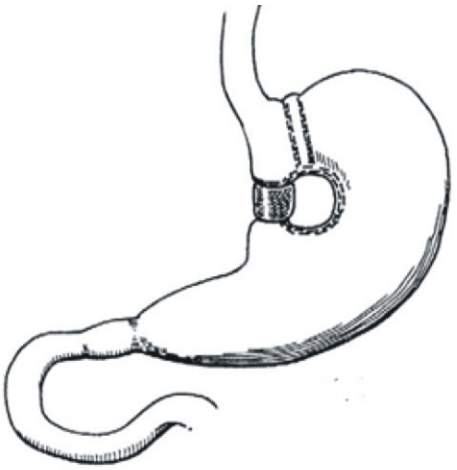


Diagram is adapted from Benjamin et al[25]

months and 36-87% in 24 months[37]. There is also improvement in co morbidities like asthma, triglyceridaemia, hypertension and type 2 diabetes[38].

Vertical Banded Gastroplasty

In this procedure, the stomach is portioned with a stapling device to create a very small proximal pouch. The stoma is then reinforced between the pouch and the gastric remnant with a mesh band thereby, limiting the passage of food into the body of the stomoach[39]. Filling of the upper stomach pouch results in satiety, and decreased solid food consumption leads to weight loss.

Complications following this procedure include staple-line dehiscence and stomal stenosis[40]. The inferior results of this

Figure 4: Sleeve Gastrectomy

Diagram is adapted from Benjamin et al[25]

procedure when compared to other restrictive procedures in terms of weight loss and resolution of co morbidities has led to its disfavour among most surgeons[41].

Sleeve Gastrectomy

This is a relatively new procedure originally published by Marceau et al[42]. This operation can be performed by either open or laparoscopic method. It is a restrictive surgery in which about 85% of the stomach is removed leaving a cylindrical or sleeve-shaped stomach with a capacity ranging from 60-150cc. The stomach is reduced to about 15% of its original size by surgical removal of a large portion of the stomach along the greater curvature. The open edges are then attached together with the help of stapler to form a sleeve or tube with a banana shape. It permanently reduces the size of the stomach. Following this procedure, there is considerable reduction in the quantity of food eaten and there is virtual elimination of the hormone (Ghrelin) which stimulate hunger[43].

Complication rate is small, about 0.5-1% and may include leakage along the staple line and post operative haemorrhage[43].

MALABSORPTIVE PROCEDURES

These procedures induce decrease small intestinal absorption of nutrients calories by bypassing intestinal loops. This may lead to short bowel syndrome which results in negative energy balance and weight loss. Excessive protein calorie malnutrition and macro and micro nutrient deficiency often accompany the weight loss caused by malabsorption. These metabolic side effects have led to the limitation of the wide use of these procedures[25]. The common malabsorptive procedures are Jejuno ileal Bypass and Bilio Pancreatic Division and Duodenal Switch. (Figure 5 — 6)

Figure 5: Jejunoileal Bypass

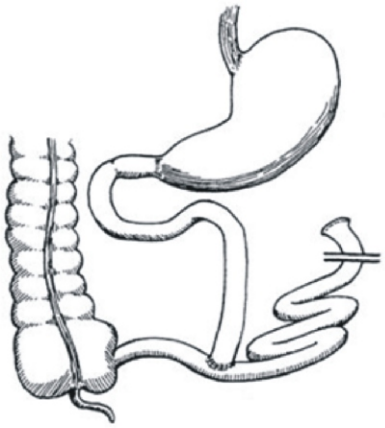


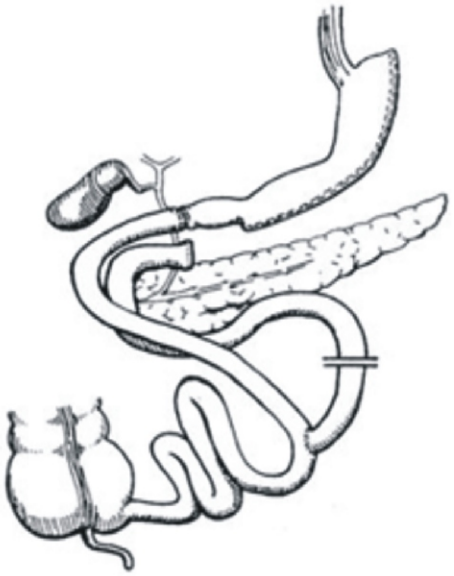
Diagram is adapted from Benjamin et al[25]

Jejunoileal Bypass

This was the first operation designed for weight loss[44]. This involves dividing the proximal jejunum, which is then attached to the ileum, but proximal to the ileocaecal valve thereby, bypassing most of the absorptive small intestine.

In spite of the satisfactory weight loss accompanying this procedure, it was abandoned because of the associated complications, most of which arose from blind loop syndrome. The complications may include oxalate kidney stones, polyathralgia, cirrhosis, liver failure, metabolic derangements (metabolic bone disease, vitamin B12 and D deficiency) that required surgical revision or reversal[45].

Figure 6 Biliopancreatic Diversion and Duodenal Switch



Adapted from Benjamin et al[25]

Biliopancreatic Diversion and Duodenal Switch

These procedures were developed to avoid the complications of blind loop syndrome. The biliopancreatic diversion involves a distal (80%) gastrectomy and a roux-en-y reconstruction consisting of a 200cm alimentary (food limb, a long biliopancreatic limb and a 50cm common limb[46]. The duodenal switch is a similar procedure but also includes a sleeve gastrectomy and duodenoileostomy to avoid complications of marginal ulcer often seen in biliopancreatic diversion[47]. In spite of the improvement in co morbid conditions and good long term weight loss following these procedures, long term complications of protein calorie malnutrition, vitamin deficiencies, metabolic bone disease and hepatic dysfunction are still major limitations[48].

CONCLUSION

The management of obesity is complex, requiring a multidisciplinary approach involving the physicians, psychiatrists, dieticians, counsellors and surgeons. Research shows that morbid obesity is a disease that remains generally unresponsive to diet and drug therapy.

Surgery is the most effective treatment for morbid obesity producing long lasting weight loss and improvement or remission of co morbid conditions. Specific criteria must be met and the patient motivated and fully informed. The type of surgery offered depends on the patient, associated co morbidities and the experience of the surgeon. Restrictive procedures are more commonly performed because they are simpler to perform and achieve good weight loss while avoiding the metabolic complications associated with malabsorptive procedures. However, life long support and behavioural modification are necessary for long term success.

REFERENCES

1. Monkhouse SJW, Morgan JDT, Bates SE, Norton SA. An overview of the surgical management of morbid

obesity. Postgrad Med J 2009; 85: 678-681
2. Mokdad AH, Bowman BA, Dietz WH, Vinicor F, Bales VS, Marks JS. Prevalence of obesity, diabetes and obesity-related health risk factors. JAMA 2003;289:76-79
3. Mokdad AH, Bowman BA, Ford ES, Vinicor F, Koplan JP. The continuing epidemics of obesity and diabetes in the United States. JAMA 2001;286:1195-2000
4. Sturm R. Increases in clinically severe obesity in the United States. Arch Intern med 2003;163:2146-2148
5. Fleqal KM, Carroll MD, Oaden CL, Johnson CL. Prevalence and trends in obesity among US adults. JAMA 2002;288:1530-1538
6. Allison DB, Fortaine KR, Manson IE, Stevens I, Vanltallie TB. Annual deaths attributable to obesity in the United States. JAMA 1999;282:1530-1538
7. Liu IH, Zinamond D, Etzioni DA, O'connell IB, Maggard MA, Livingstone EH et al. Characterizing the performance and outcomes of obesity surgery in California. Am Surg 2003;69:823-828
8. Akinkugbe OO. Non- communicable diseases in Nigeria, the next epidemics: Nigeria preparedness. Third Biennial Abayomi Bamidele Memorial Lectures. Niger J Clin Pract 2000; 3: 37-42
9. Simimialayi IM, Emem-Chioma PC,Dappa DV. The prevalence ofobesity as indicated by BMI and waist circumference among Nigerian adults attending clinics as out patients in Rivers State. Niger J Med 2008; 17:340-345
10. Flier JS, Maratos-Flier E. In Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, Loscalzo J (eds). Harrison's Principles of Internal Medicine, 17th edition. New York. Mc Graw Hill 2008; 444-447
11. Inni A, Asakawa A, Bowers CY. Ghrelin, apetite and gastric motility: the emerging role of the stomach as an endocrine organ. FASEB J 2004;18:439-456
12. Lall S, Tung LY, Ohlsson C, Jansson JO, Dickson SL. Growth hormone-independent stimulation of adiposity by Growth hormone secretagogues. Biochem Biophys Res Common 2001:280:132-138
13. Hewson AL, Dickson SL. Systemic administration of ghrelin induces Fos and Egr-1 proteins in the hypothalamic arcuate nucleus of fasted and fed rats. J Neuroendocrinol 2000;12:1047-1049
14. Dickson SL, Lukman SM. Induction of C-Fos messenger ribonucleic acid in neuropeptide Y and growth hormone releasing factor neurons in the rat arcuate nucleus following systematic injection of the growth hormone secretagogue, growth hormone releasing peptide-6. Endocrinology 1997;138:771-777
15. Jerlhag E, Egecioglu E, Dickson SL, Andersson M, Svensson L, Engel JA. Ghrelin stimulates locomotor activity and Accumbal Dopamine-overflow via central cholinergic systems in mice: implications for its involvement in Brain Reward Addiction. Biology 2004;11:45-54
16. Elia M. In Kumar P, Clark M (eds). Clinical Medicine, 7th edition. Spain. Saunders 2009; 228
17. Bray GA. Medical consequences of obesity. J Clin Endocrinol Metab 2004;89:2583-2589
18. Probert C, Monkhouse SJW, Morgan JDT, Norton DA. Obesity Surgery. In: Probert C(ed). Recent advances in gastroenterology 11 London: Royal society of medicine press Ltd,2009:75-93
19. Goodrick GK, Foreyt JT, Renquist KE. A decade of change in obesity surgery. Obes Surg 1997;7:189-197
20. Monkhouse SJW, Morgan JDT, Bates SE, Norton SA. A overview of the management of morbid obesity. Postgrad Med J 2009;85:678-681
21. O'Brien PE, Brown WA, Dixon JB. Obesity, weight loss and bariatric surgery. Med J Aust 2005;183:10-14
22. SAGES Guidelines Committee. SEGES guidelines for clinical application of laparoscopic bariatric surgery. Surg Endosc 2008;22:2281-2300
23. Di Cosmo L, Vuolo G, Piccolomini A. Bariatric surgery: early results with a multidisciplinary team. Obes Surg 2000;10:272-273
24. National Institute for Health and Clinical Excellence. CG43 obesity guidelines, December 2006
25. Benjamin E, Schneider MD, Edward C, Mun MD. Surgical management of morbid obesity. Diabetes Care 2005;28:475-480
26. Kellom M, Kuemmerle JF, O'Dorisio TM, Rayford P, Martin D, Engle K et al. Gastrointestinal hormone responses to meals before and after gastric bypass
27. Wittgrove AC, Clark GW, Temblay LJ. Laparoscopic gastric bypass, Roux-en-y: preliminary report of 5 cases. Obes Surg 1994;4:353-357
28. Tritos N, Mun EC, A B, Grayson R, Maratos-Flier E, Goldfine A, Serum ghrelin levels in response to glucose load in obese subjects post gastric bypass surgery. Obes Res 2003;8:919-9
29. Mun EC, Blackburn GL, Matthew JB. Current status of medical and surgical therapy for obesity. Gastroenterol 2001;120:669-681
30. Schaur P, Ikramuddin S, Hamad G,Gourash W. The learning curve for laparoscopic Roux-en-y gastric bypass of 100 cases. Surg Endosc 2003;17:212-215
31. Pories WJ, Swanson MS, MacDonald KG, Long SB, Morris PG, Brown BM et al. Who would have thought it? An operation proves to be the most effective therapy for adult-onset diabetes mellitus. Ann Surg 1995;222:339-350
32. Schauer PR, Burguera B, Ikramuddin S, Cottam D, Gourash W, Hamad G, et al. Effect of laparoscopic roux-en-y gastric bypass on type 2 diabetes mellitus. Ann Surg 2003;238:467-484
33. Wittgrove AC, Clark GW. Laparoscopic gastric bypass, roux-en-y: 500 patients: technique and results with 3-60 month follow-up. Obes Surg 2000;10:233-239
34. Rubestein R. Laparoscopic adjustable gastric banding at a US center with up to 3 year follow-up. Obes Surg 2002;12:380-384
35. O'Brien PE, Dixon JB, Brown W, Schader LM, Chapman L, Burn AJ, et al. The laparoscopic adjustable gastric band: a prospective study of medium- term effects on weight, health and quality of life. Obes Surg 2002;12:652-660
36. O'Brien PE, Dixon JB. Weight loss and early and late complications: the international experience. Am J Surg 2002;184:42S-45S
37. DeMaria EJ, Surgerman HJ, Meador JG, Doty JM, Kellum JM, Wolfe L, et al. High failure rate after

laparoscopic adjustable gastric silicon banding for treatment of morbid obesity. Ann Surg 2001;233:809-818
38. Fauretti F, Cadiere GB, Segato G, Himpens J, DeLuca M, Busetto L, et al. Laparoscopic banding: selection and technique in 830 patients. Obes Surg 2002;12:385-390
39. Mason EE. Vertical banded gastroplasty for obesity. Arch Surg 1982;117:701-706
40. Nightengale MC, Sarr MG, Kelly KA, Jensen MD, Zinsmeister AR, Palumbo PJ. Prospective evaluation of vertical banded gastroplasty as the primary operation for morbid obesity. Mayo Clinic Proc 1991;66:773-782
41. Sugerman HJ, Londrey GL, Kellum JM, Wolf L, Liszka T, Engle KM, et al. Weight loss with veftical banded gastroplasty and roux-en-y gastric bypass for morbid obesity with selective versus random assignment. Am J Surg 1989;157:93-102
42. Masceau P, Biron S, Borque RA. Bilopancreatic diversion with a new type of gastrectomy. Obes Surg 1993;3:29-35
43. Mishra RK. Text book of Practical Laparoscopic Surgery(2nd edition),2009. Jaypee, New Delhi, 267
44. Payne JH, Dewind LT. Surgical treatment of obesity. Am J Surg 1969;118:141-147
45. Deitel M, Shahi B, An and Pk, Deitel FH, Cardinell DL. Long-term outcome in a series of jejunoileal bypass patients. Obes Surg 1993;3:247-252
46. Scopinaro N, Gianetta E, Adami GE, Friedman D,