# Outcome of Abdominoplasty after one year post-operative

Salih Al-Baaj Plastic surgeon F.I.C.M.S, F.A.C.S Diwaniyah Teaching Hospital

# Abstract

# **Background** :

The majority of patients that have been underwent abdominoplasty show lasting weight loss after one year. This long term weight loss may be related to many factors, but the most obvious cause is the increase in satiety feelings full after eating. Many questions remain controversial about the permanent weight reduction.

### Methods :

A retrospective study included those patients underwent abdominoplasty and we attempted to determine the most factor associated with weight loss. This study started between April 1999 to June 2010, it includes 40 patients underwent abdominoplasty at general and private hospitals. From the original 40 patients, 18 could not be contact for the follow up interview and thus were excluded from the results of the study. Other 2 patients were also excluded from the analysis because of their pregnancy in the postoperative years. Of the remaining 20 patients (4 males and 16 females), from them 2 patients had previously underwent bariatric surgery but those were included in our results. Those 20 patients ranged in age from 25 to 55 years.

# **Results:**

All patients in this study that undergoing abdominoplasty had weight loss beyond that of their resected pannus, with minimum body mass index reach (18 + 2 weeks) after surgery. The weight loss is due to the increase in satiety by 75% (number = 15). A preoperative basal metabolic index greater than 24.5 K/m<sup>2</sup> can be used to predict the long term weight loss with sensitivity and specificity 95% and 85% respectively.

# **Conclusion:**

Obese patients performed abdominoplasty with basal metabolic index greater than 24.5 Kg/m<sup>2</sup>, appear to be more liable to lose weight after one year from the time of the operation. This is due to the increased satiety seen in many our patients is related to the changes in the neuroendocrine system. The removal of fat cells from the abdomen may leads to reduce the level of the hormones affecting appetite, which are secreted by fatty tissues. Sustained weight loss was also related more likely to greater amount of excess abdominal tissue removed.

### Introduction:

The prevalence of obesity, defined by the National Institutes of Health as BMI of 30 Kg /  $m^2$ [1] or greater has more than double in the US

since 1980 **[2]**. According to a recent National Health and National Examination Survey, a staggering 33.3 percentage of adult men and 35.3 percent of adult woman in the United

States are currently considered obese [2]. Obesity is caused by a long term positive energy balance where intake is greater than expenditure, with portion sizes and consumption of high-calorie foods continuing to increase and physical activity on the decline, it is not surprising that the rate of obesity continues to decline. The obesity epidemic presents a major health concern, as it increases the risk of many diseases and health conditions, including but not limited to sleep apnea, diabetes mellitus, hypertension, osteoarthritis, dysIipidemia, and certain types of cancers, gall bladder disease, stroke, and coronary heart disease [3]. Not only obesity is associated with serious comorbidities, it is also very costly [4]. Despite the growing need for the therapeutic strategies to achieve and maintain weight loss, such treatment remain Iimited [5]. Bariatric surgical procedure, such as gastric bypass are among the few current treatment that produce permanent weight Ioss [6]. Despite its efficacy, surgical treatment of obesity has general been to the patients suffering from morbid obesity, those with a body mass index of 40kg/m2 or greater, as the majority of these patients would gain more benefit as compared with their overweight counterparts. Surgical treatment is now considered the most effective treatment for morbidIy obese population [7]. Most evidence points to decrease morbidity and mortality as a result of the significant weight loss associated with this surgery [8,9]. Abdominoplasty is a surgical treatment available to a greater spectrum of patients looking to decrease the size and improve the aesthetics of their midsection, whether because of a Iarge abdominal pannus from massive weight resulting Ioss demachalasis, and prominent streia following multiple pregnancies, or because of scarring and hernia formation produced by previous operation [10]. Whether or not long-term weight reduction is associated with AbdominopIasty

has been Iittle investigated and remains controversial [11]. Possible factors implicated with weight Ioss following abdominopIasty include technique premorbid weight, motivation, postoperative diet and excises, previous bariatric surgery, and size of pannus resected. The purpose of the presented study was to determine whether or not our patient population was successful in obtaining a weight reduction after abdominopIasty, and if so, what factors were associated with maintaining Iongterm with weight reduction.

# **Patients and Methods:**

This study included 40 patients underwent abdominoplasty that were performed by one surgeon at private and general hospitals. It is started Between April 1999 and June 2010. A retrospective study; it include a chart review and in-depth patient follow up interview were conducted on these patients to obtain the following informations: age, height, sex of the patients, weight before operation, weight of the resected pannus, previous bariatric surgery, life postoperative style, (diet, regimen, exercise), and satisfaction with abdominoplasty results. From the original 40 patients, 18 patients could not be contact for follow up interview, so those were excluded from the study. Other

Patients were excluded from the analysis because of their pregnancy after postoperative years.

So the remaining were 20 patients (4 males and 16 females), from these patients two had underwent previously bariatric surgery, but those included with the results. The age of these patients were ranged from 25 to 55 years.

# Surgical technique:

The surgical technique used involved pannus resection as in Jack-knife 90 degree – flexion

| AL-Qadisiyah Medical Journal | Vol.15 | No.1 |
|------------------------------|--------|------|
|------------------------------|--------|------|

July 2019

position, which was preceded by a tight twolayer permanent suture rectus anterior fascia plication from the xiphoid to the pubic bone (**Figure 1 and 2**). Pre and postoperative prophylactic anticoagulant (Enoxaparen 2000 IU) was given. The patient encouraged to exercise for at least 1 hour per week, starting 8 weeks after surgery, and also we advise them to eat healthy diet (**Figure 3**). This analysis included to calculate the changes in the body weight and the mass index after operation by using the patient's postoperative body mass index and calculate the preoperative weight minus the weight of the resected pannus as the baseline body mass index. We use other data that the weight loss lasting less than one year consider as a short term weight loss, where the weight loss enduring more than one year was considered as a long term weight loss.

Figure (2): the surgical technique:

# 

# Figure (1): the surgical technique:

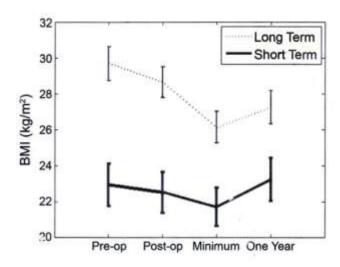
**Results:** 

| AL-Qadisiyah Medical Journal | Vol.15 | No.1 | July 2019 |
|------------------------------|--------|------|-----------|
|------------------------------|--------|------|-----------|

According the receiver operating to curve characteristic analysis, the high sensitivity and specificity cut off point for long term. Of the patients with basal metabolic index above 24.5 Kg/m<sup>2</sup>, 43% (number =13) have long term weight loss at one year as compared with 17% (number = 1) of patients below this threshold. The trend of the short and Iong - term weight Ioss group are shown for preoperative BMI. postoperative BMI, minimum BMI, and 1 year BMI. (Figure 4).

The charted informations were obtained on these 20 patients with an average follow up interview time of 36 month. This chart included the preoperative mean body index 28  $\pm$  1.0 Kg/m<sup>2</sup> and the weight of the resected pannus was 6 $\pm$  0.5 Kg. In this this analysis of the 20 patients 14 (70%) have sustained weight loss beyond of their postoperative weight. That means (preoperative weight loss minus pannus resected after one year). The remaining 6 patients (30%) experience some degree of weight loss after one year of their following surgery. Those patients with long weight loss, the preoperative body mass index was 30  $\pm$  0.5 Kg/m<sup>2</sup> versus 22  $\pm$  1.5 Kg/m<sup>2</sup>.

Figure (4): pre-operative, post-operative, minimum and after one year weight loss in relation to BMI



The short and Iong – term data are significantIy different for aII four time points. Both the maximum change in BMI and the charge in BMI at 1 year were not significantIy different between the two pannus groups. When patients were asked for the most important factors contributing to their weight Ioss, 75% (n=15) reported an increased feeIing of satiety, either with eating or generaIIy throughout the day. When asked what Ied to weight Ioss , onIy one (5%) attributed it to diet aIone , eight (40)

percent ) to satiety alone ; five ( 25 percent ) to combination of diet , exercise, and /or satiety ; three ( 15 percent ) to their previous gastric bypass , and the remaining three (15 percent ) to other reasons of those experiencing satiety , 60 percent ( n=9) retained that sensation at 1 year, whereas in other 40 percent (n=6) it Iasted an average of 39 months.

For the long-term weight loss group specifically, 85 percent (n=12) reported a change in satiety (seven had early satiety with

No.1

have less body fat to be lost. So it possible that

eating only and have five had general feeling of fullness at all times). For the short-term weight loss group 50 percent (n=3) reported an increase in satiety with a majority of those having a general sense of satiety throughout the day.

# **Discussion:**

Our study is retrospective, it include 20 patients undergoing abdominoplasty, and all of the patients had some degree of weight loss after one vear. The most commonly factor responsible for this weight loss is a sense of satiety, that is found in 75% (number = 15) of the patients, this is either as general satiety throughout the day or early satiety with eating. Most of the patients 60% (number = 9) show experiencing satiety maintain that sensation at one year, where in other, it lasted in average of  $(14.6 \pm 2.8 \text{ weeks})$ . It is possible that the sensation of satiety is lost or decreased which was a contributing factor to the weight loss. To determine what are the factors which were responsible to the long term loss, we found that the patient with preoperative body mass index greater than or up to 24.5 Kg/m<sup>2</sup> achieved long term weight loss with sensitivity and specificity 93% and 84% respectively. So the body mass index is significantly correlated. The patient with preoperative body mass index greater than or equal to 24.5 Kg/m<sup>2</sup>, those patients with percentage 93% (number = 13) showed maintained long term weight loss after one year. As compared with 17% (number = 1) of those below this threshold. After one year some patients below this threshold have gained an average of  $2.0 \pm 1.5\%$  in those with body mass index above 24.5 kg/m<sup>2</sup>. This study tell us that overweight obese patients tend to have more long term weight loss benefit and weight reduction from the normal weight counterparts. This may be because the normal weight patients any changes in the neuroendocrine factors affected the overweight and the obese patients to a greater extent. A second factor significantly correlated with long term weight loss is the weight of the resected pannus, those patients with pannus resection weighting greater than 5 kg had significant changes in the body mass index after one year time points (P=0.029 and P=0.01 respectively) compare with those with small resection. It mean that the greater the amount of the fat cells removed, the greater the impact on the neuroendocrine system. Milieu regulating satiety and weight loss balance as discussed below in particularly the possibility that removing fat cells that produce leptin hormone may reduce leptin resistance has been described in the obese patients [12]. We hypothesize that the increased satiety seen in our patient and subsequent weight loss is related to change in the neuroendocrine system. This is supported by the Iatest studies on appetite that have found that food intake is regulated by the action of gastrointestinal peptide hormones and Ieptin, hormones secreted from adipose tissue, on the central nervous system . These hormones act as satiety signals in the vagal-brainstemhypothaIamic pathway [13]. In the hypothalamus, gut hormones and leptins act by stimulating inhibiting neurons in the arcuate nucleus of the hypothalamus. In turn, this control center responds by expressing peptides that either stimulate or inhibit food intake [13,16]. Afferent signals from the vagus nerve convey information about the mechanical and chemical stimulation of the gastrointestinal tract by ingested food to the brainstem . This further elicits refiexes that control gastrointestinal functions and sends signals to the hypothalamus to inhibit food intake [16-17]. The vagus nerve contains mechanoreceptor that are sensitive to stomach and intestinal volume and luminal pressure and receptors for a number of gut hormones. This then conveys information about the ingested food [16,18,21]. Studies have shown that vagotomy abolishes the appetite – modifying actions of these gut hormones [20,22,24].

Many gastrointestinal peptide hormones and Ieptins have been discovered. They are known to inhibit / stimulate food intake by acting at the vagus nerve and /or the arcuate nucleus. For example, leptin, which is released from adipose tissue, and insuIin, which is secreted by the pancreas, both function within the hypothalamus to inhibit food intake [15,25,26]. Cholecystokinin, in contracts, is secreted by the L cells of the small intestine and acts mainly through the vagus nerve to the inhibit feeding by modifying gastrointestinal tract functions [15,20,27,28]. Peptide YY, oxyntomodulin and glucagon-like peptide -1 are all secreted from I ceIIs in the intestines. They act on both the hypothalamus and the vagus nerve to inhibit food intake [15,28,29,30]. Pancreatic peptide, from the pancreas ; glucose released dependent insulinotropic polypeptide, secreted from the stomach, duodenum, and jejunum; adiponectin, produced by adipose tissue; and amylin, from the pancreas, are other satiety promoting gut hormones [13,15,16]. So far, ghreIin, which is produced in the stomach, is the only known circulating appetite stimulant. It functions at the hypothalamic level. [13,15,16]. This is a pilot study, and with future studies, we would propose measuring levels of these gastrointestinal peptide hormones before our patients undergo abdominopalsty and then at incremental times after surgery. By comparing hormone IeveIs before and after abdominopIasty, we will be able to determine whether there is any significant change in gastrointestinal peptide hormone or leptin expression. This will elucidate the satiety signals that are responsible for loss of appetite found in our patients. Further studies will

clarify the mechanisms of appetite regulation and may lead to the creation of an injectable appetite suppressant drug. With the increasing global prevalence of obesity and its ensuring physiologic, psychological, and economic implications, the need to understand appetite control is imperative.

# **Conclusion:**

Obese patients performed abdominoplasty with basal metabolic index greater than 24.5 Kg/m<sup>2</sup>, appear to be more liable to lose weight after one year from the time of the operation. This is due to the increased satiety seen in many our patients is related to the changes in the neuroendocrine system. The removal of fat cells from the abdomen may leads to reduce the level of the hormones affecting appetite, which are secreted by fatty tissues. Sustained weight loss was also related more likely to greater amount of excess abdominal tissue removed.

### **References:**

1. National Institutes of Health, National Heart, I ung, and Blood Institute. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidenc e Report. Washington, DC: US . Department of Health and Human Services, Public Health Servi ce, National Institutes of Health, National Heart, Iung, and Blood Institute; 1998.

Cited Here...

2. Ogden CI, CarroII MD, McDoweII MA, fiegaI KM. Obesity among adults in the United States: No statistically significant change since 2003-2004. NCHS Data Brief 2007;1:1-8.

Cited Here...

3. Pi-

Sunyer FX. Comorbidities of overweight and ob esity: Current evidence and research issues. Med Sci Sports Exerc. 1999;31:5602-

5608. Cited Here...

4. finkeIstein EA, fiebeIkorn IC, Wang G. Nation aI medicaI spending attributable to overweight a nd obesity: How much, and who's paying? HeaIt h Affairs (MiIIwood) 2003;Supp1 Web Exclusiv es:W3-219-W3-226. Cited Here... 5. Bray GA, Tartaglia IA. Medicinal strategies in

the treatment of obesity. Nature 2000;404:672-677.

Cited Here...

6. Sjostrom I. Surgical intervention as a strategy for treatment of obesity. Endocrine 2000;13:213-230.

Cited Here...

7. Iivingston EH. Obesity, mortality, and bariatri c surgery death rates. JAMA 2007;298:2406-2408.

Cited Here...

8. Buchwald H, Avidor Y, Braunwald E, et al.. B ariatric surgery: A systemic review and metaanalysis. JAMA 2004;292:1724-

1728. Cited Here...

9. Christou NV, SampaIis JS, Iiberman M, et al.. Surgery decreases Iong-

term mortality, morbidity, and health care use in morbidIy obese patients. Ann Surg. 2004;240:41 6-423; discussion 423-424.

Cited Here...

10. Savage RC. AbdominopIasty foIIowing gastr ointestinal bypass surgery. Plast Reconstr Surg. 1983;71:500-509.

Cited Here...

11. Shermak MA, Bluebond-

Iangner R, Chang D. Maintenance of weight Ioss after body contouring surgery for massive weigh t Ioss. Plast Reconstr Surg. 2008;121:2114-2119.

Cited Here...

12. EI-Haschimi K, Iehnert H. Ieptin-resistance--Or why Ieptin fails to work in obesity. Exp Clin E ndocrinoI Diabetes 2003;111:2-7.

F

13. Chaudhri OB, SaIem V, Murphy KG, BIoom SR. Gastrointestinal satiety signals. Annu Rev P hysioI. 2008;70:239-255.

Cited Here...

14. Cone RD, Cowley MA, Butler AA, Fan W,

Marks DI, Iow MJ. The arcuate nucleus as a con duit for diverse signals relevant to energy homeo stasis. Int J Obes Relat Metab Disord. 2001;25:S 63-S67.

Cited Here...

15. Austin J, Marks D. Hormonal regulators of a ppetite. Int J Pediatr EndocrinoI. 2009;2009:141 753.

Cited Here...

16. Strader AD, Woods SC. Gastrointestinal hor mones and food intake. Gastroenterology 2005;1 28:175-191.

Cited Here...

17. Moran TH, Iadenheim EE, Schwartz GJ. Wit hin-

meaI gut feedback signaIing. Int J Obes ReIat M etab Dis. 2001;25(Supp15):S39-

S41. Cited Here...

18. Berthoud HR. Multiple neural systems contr oIIing food intake and body weight. Neurosci Bi obehav Rev. 2002;26:393-428.

Cited Here...

19. Powley TI, Phillips RJ. Musings on the wand erer: What's new in our understanding of vago-

vagaI refiexes? I. Morphology and topography of vagaI afferents innervating the GI tract. Am J Ph ysioI Gastrointest Iiver PhysioI. 2002;283:G121 7-G1225.

Cited Here...

20. Moran TH, Kinzig KP. Gastrointestinal satie ty signals II. Cholecystokinin. Am J Physiol Gas trointest liver Physiol. 2004;286:G183--G188. Cited Here...

21. Date Y, Murakami N, Toshinai K, et al.. The role of the gastric afferent vagal nerve in ghrelin

induced feeding and growth hormone secretion i n rats. Gastroenterology 2002;123:1120-1128. Cited Here...

22. Abbott CR, Monteiro M, SmaII CJ, et a1.. Th e inhibitory effects of peripheral administration of peptide YY(3-36) and glucagon-like peptide-1 on food intake are attenuated by ablation of the vagaI-brainstem-

hypothaIamic pathway. Brain Res. 2005;1044:1 27-131. Cited Here...

23. Koda S, Date Y, Murakami N, et al.. The role of the vagal nerve in peripheral PYY3-36-induced feeding reduction in rats. Endocrinology

2005;146:2369-2375.

Cited Here...

24. Asakawa A, Inui A, Yusuriha H, et al.. Chara cterization of the effects of pancreatic polypeptid e in the regulation of energy balance. Gastroente rology 2003;124:1325-1336.

| AL-Qadisiv | ah Medical Journal | Vo |
|------------|--------------------|----|
| AL-Quuisi  | an medical Journal |    |

ol.15 No.1

Cited Here...

25. Elias CF, Aschkenasi C, Iee C, et al.. Ieptin d ifferentidIIy regulates NPY and POMC neurons projecting to the Iateral hypothalamic area. Neur on 1999;23:775-786.

Cited Here...

26. Cheatham B, Kahn CR. InsuIin action and th e insuIin signaIing network. Endocr Rev. 1995;1 6:117-142.

Cited Here...

27. Druce M, BIoom SR. The regulation of appet ite. Arch Dis Child. 2006;91:183-187.

Cited Here...

28. D'Alessio D. Intestinal hormones and regulat ion of satiety: The case for CCK, GIP-

1, PYY, and Apo A-

IV. JPEN J Parenter Enteral Nutr. 2008;32:567-568.

Cited Here...

29. Batterham RI, Cowley MA, Small CJ, et al.. Gut hormone PYY(3-

36) physiologically inhibits food intake. Nature 2002;418:650-654.

Cited Here...

30. Baggio II, Huang Q, Brown TJ, Drucker DJ.

Oxyntomodulin and glucagon-like peptide-

1 differentially regulate murine food intake and energy expenditure. Gastroenterology 2004;127: 546-558.

Cited Here...