INTRODUCTION

Obesity is a major health problem worldwide and it becomes an epidemic disease which is defined as a complex disorder involving an excessive amount of body fat obesity is related mainly to body mass index which is the weight in kilograms divided by the square of height in meters. Morbid obesity is defined when body mass index equals or more than 40 kg/m^2 ***(Steinberger et al., 2009).***

Obesity causes many pathophysiological changes on body systems. On respiratory system cause reduced lung volumes, decreased lung compliance, obstructive sleep apnea and abnormal ventilation pattern. Cardiovascular diseases dominate the mortality and morbidity of obese people as obesity by increasing incidence of atherosclerosis, hypertension, thromboembolic diseases, ischemic heart diseases and cardiac failure.Obese patient have a high risk of type 2 diabetes mellitus, fatty liver diseases, gall bladder diseases, osteoarthiritis and social disapproval ***(Zimmet et al., 2007).***

Bariatric surgical procedures are an effective treatment for achieving weight loss especially in case of medical treatment failure. The most commonly performed bariatric surgical procedures are laparoscopic adjustable gastric banding (LAGB), ROUX-EN-Y gastric bypass, sleeve gastrectomy, gastric plication and gastroplasty ***(Karamanakos et al., 2008).***

Many challenges face anesthesiologists in anesthetic management of obese patient due to pathophysiological changes on body systems so preoperative assessment facilitate the appropriate selection of patient for surgery. Due to respiratory system changes obese patient show many problems at induction of anesthesia as severe hypoxia even after short period of apnea so care must be taken during induction with adequate preoxygenation and PEEP use in mask ventilation period. Obese patient who has clinically significant obstructive sleep apnea may be predisposing to airway difficulties during anesthesia as severe hypoxia and postoperative pulmonary complications. Position of patient has an important role in reducing postoperative complications as being in ahead elevated laryngoscopy position during operation show adecrease in post intubation atelectasis ***(Chalhoub et al., 2007).***

Cardiac functions of obese patient may be worsen during anesthesia as morbid obesity is a major independent risk factor for deep venous thrombosis and postoperative pulmonary embolism causing sudden death so preoperative low molecular weight heparin subcutaneous injection is used as prophylaxis. Acid aspiration prophylaxis should be done due to high incidence of postoperative nausea and vomiting ***(Barash et al., 2006).***

Non alcoholic fatty liver diseases are seen in obese patients which may contribute with capacity of liver to metabolize drugs so anesthetic drugs must be dosed according to lean body weight not actual body weight ***(Barash et al., 2006).***

Pain management is a part of anesthesia. Opioids is commonly used to achieve analgesia. Patients will experience at least one adverse effect of opioids after surgery as itching, nausea, vomiting, muscle weakness, dizziness and hyperalgesia. In addition to this adverse effects opioids has another effects on obese patient as it causes increasing incidence of post-operative pulmonary morbidity such as respiratory depression, sedation and impending enhanced recovery after surgery which probably lead to occurrence of post-operative pulmonary embolism and deep venous thrombosis ***(Hofer et al., 2005).***

Opioid free anesthesia is done to overcome opioids complications and achieving enhanced recovery after surgery as it is important to obese patient to be full awake, pain free and mobilize early to avoid respiratory depression, atelectasis and pulmonary infection. Opioid free anesthesia improve wound healing by decreased immunosuppression effect of opioids also enhance bowel function in postoperative period ***(Mulier et al., 2013).***

Opioid free anesthesia is achieved by direct sympathetic block centrally and peripherally by using clonidine, dexmedetomidine. Indirect block of sympathetic effects by lidocaine, Mg sulfate and inhalation vapor. Multimodal analgesics loading up preoperative to be active when patient walk up by using low dose of ketamine, lidocaine, diclofenac and paracetamol ***(Mulier et al., 2013).***

AIM OF THE WORK

The aim of this work is to study the anesthetic management of obese patient during bariatric surgery without using opioids.

PHYSIOLOGICAL AND PATHOLOGICAL CHANGES OF OBESITY

Obesity

Obesity is a chronic, relapsing, stigmatized, neurochemical disease that is more prevalent in developing and developed countries leading to much comorbidity.multiple factors are involved that contribute to the development of obesity.these may be social, behavioral, environmental and genetic.it is a global health problem in the present era ***(Chen h et al., 2006)***.

Definition of obesity

Obesity definedas a chronic disease characterized by pathophysiological processes that result in increased adipose tissue mass and which can result in increased morbidity and mortality. In an environment that interacts with susceptibility genes to promote weight gain (i.e., obesogenic), many individuals have a body mass index (BMI) $\geq$ 25 kg/ m^2, which is associated with increased likelihood for obesity-related complications and risk of progressive obesity ***(Garber et al., 2013)***.

The body mass index (BMI) is the accepted standard measure of overweight and obesity for children two years of age and older. Body mass index provides a guideline for weight in relation to height and is equal to the body weight divided by the height squared (table 1).

$$BMI = \frac{\text{body weight (kg)}}{\text{height}^2\text{ (m)}}$$

Other measures of obesity, including weight-for-height and measures of regional fat distribution (eg, waist circumference and waist-to-hip ratio) are discussed separately. Adults with a BMI between 25 and 30 are considered overweight; those with a BMI ≥30 are considered to be obese ***(Baker et al., 2005)***.

Table (1): Classification of weight categories using the body mass index (BMI)

Category	BMI Level
Underweight	Less than 18.5
Normal	18.5-24.9
Overweight or pre-obese	25.0-29.9
Obese	30 and over

World Health Organisation, Obesity Factsheet,
http://www.who.int/mediacentre/factsheets/fs311/en/

Obesity and *morbid obesity* are BMI >30 and 40 kg/ m^2, respectively, while BMI > 55kg/m^2 denotes *super morbid*

obesity. BMI differentiates obese from no obese adults and it reliably measures body fat because it adjusts for height while strongly correlating with body weight; however, it cannot distinguish between overweight and over fat as heavily muscled people can be easily classified as overweight using BMI. Other factors such as age and fat distribution should therefore be taken into consideration, among other health risk predictors that utilize the concept of BMI. Body circumference indices such as waist circumference, waist-to-height ratio, and waist-to-hip ratio can identify patterns of obesity and correlate strongly with mortality and the risk for developing obesity-related diseases. Waist circumference strongly correlates with abdominal fat and is an independent risk predictor of disease. A waist circumference exceeding 102 cm (40 in) in men and 89 cm (35 in) in women indicates increased risk in overweight and obese individuals. A waist-to-hip ratio (WHR) >0.9 in women and >1.0 in men is associated with a higher risk of morbidity and mortality than a more peripheral distribution of body fat (WHR <0.75 in women and <0.85 in men) ***(Barash et al., 2006)***.

Pathogenesis of obesity

Neuroendocrine obesity

Several mechanisms lead to obesity. Obesity can follow damage to the ventromedial part of the hypothalamus in the brain, but this is rare. Cushing's disease is somewhat more common and can present with obesity. Treatment should be

directed at the cause of the increased formation of adrenal corticosteroids ***(Bray et al., 2005).***

Drug-induced weight gain

Treatment of diabetics with insulin, sulfonylureas or thiazolidinediones can increase hunger and food intake, resulting in weight gain. Treatment with some antidepressants, antiepileptics and neuroleptics can also increase body weight, as can cyproheptadine, probably through effects on the monoamines in the central nervous system ***(Bray et al., 2005)***.

Dietary obesity

Eating a high-fat diet and excessive consumption of sugar-sweetened beverages and the prevalence of abundant varieties of food in cafeterias or supermarkets are dietary factors in the development of obesity. Larger portion sizes increase the amount we eat. It would seem a simple enough behavioral change to ‘‘eat less’’, but the programs that have tried to introduce behavior change strategies have made few effective inroads yet ***(Bray et al., 2005).***

Reduced energy expenditure:

Reduced energy expenditure relative to energy intake is the other major component in the cause of obesity in modern society. Energy expenditure can be divided into four parts: resting metabolism ranges from 3.37 to 3.76 MJ/m2/24 h (800-

900 kcal/sq m/24 h). It is lower in females than in males, and declines with age. This decline with age could account for much of the increase in fat stores if food intake does not decline similarly. Physical exercise is variable but on average is responsible for about one-third of the daily energy expenditure. From a therapeutic point of view, this component of energy expenditure is most easily manipulated. Dietary thermogenesis is the energy expenditure that follows the ingestion of a meal. Heat produced by eating may dissipate up to10% of the ingested calories. Protein appears to have the largest effect. These thermic effects of food are one type of metabolic ‘‘inefficiency’’ in the body, that is, where dietary calories are not available for ‘‘useful’’ work. In the obese, the thermic effects of food are reduced particularly in individuals with impaired glucose tolerance or diabetes. Acute over- or underfeeding will produce corresponding shifts in overall metabolism, which can be as large as 15-20%. Thus increasing physical activity, and energy expenditure would seem a good way of ‘‘preventing’’ obesity ***(Bray et al., 2005).***

Genetic factors in obesity:

Syndromes of obesity:

Genetic factors can produce some types of obesity that are easily recognized. Among these type of obesity are:

- The Bardet-Biedl syndrome, characterized by retinal degeneration, mental retardation, obesity, polydactyly, and hypogonadism; (2) the Alstrom syndrome, characterized by pigmentary retinopathy, nerve deafness, obesity, and diabetes mellitus; (3) Carpenter syndrome, characterized by acrocephaly, mental retardation, hypogonadism, obesity, and preaxial syndactyly; (4) the Cohen syndrome, characterized by mental retardation, obesity, hypotonia, and characteristic facies; (5) the Prader-Willi syndrome, characterized by hypotonia, mental retardation, hypogonadism, and obesity; and (6) the pro-opiomelanocortin (POMC) syndrome, characterized by defective production of POMC that is recognized as a red-headed fat child with a as a low.
- Plasma cortisol

(Bray et al., 2005)

Genetic susceptibility to obesity

If both parents are obese, about 80% of the offspring will be obese. If only one parent is obese, the likelihood of obesity in the offspring falls to less than 40%. Studies with identical twins suggest that inheritance accounts for up to 70% and environmental factors (diet, physical inactivity, or both) account for the rest of the variation in body weight ***(Bray et al., 2005).***

Single gene causes of obesity

Leptin deficiency and deficiency of the leptin receptor are rare, but are associated with massive human obesity. The most common defects associated with massive obesity are abnormalities in the melanocortin receptor system where up to 5% of massively obese young people may have this type of defect ***(Bray et al., 2005).***

Pathophysiological changes of obesity

Metabolic Complications of Obesity

A central or upper body fat distribution, more so than total fat mass, is predictive of the metabolic complications of obesity. Adipose tissue release of free fatty acids (FFAs) and glycerol into the circulation through lipolysis provides 50 to 100% of daily energy needs. Adipose tissue lipolysis is regulated primarily by insulin (inhibition) and catecholamines (stimulation), although growth hormone and cortisol also stimulate lipolysis. Upper body obesity is associated with several abnormalities of adipose tissue lipolysis, most remarkably with higher FFA concentrations due to excess release postprandially. Abnormally high FFA concentrations can contribute to a number of the metabolic complications of obesity ***(Goldman et al., 2012).***

Insulin Resistance

- The term *insulin resistance* is typically used when referring to the ability of insulin to promote glucose uptake, oxidation, and storage as well as to inhibit the release of glucose into the circulation. The primary site of insulin stimulated glucose uptake, oxidation, and storage is skeletal muscle. The principal site of glucose production is the liver. Insulin resistance initially leads to hyperinsulinemia and may eventually lead to the development of type 2 diabetes mellitus. The ability of insulin to promote glucose uptake, oxidation, and storage in muscle and to suppress plasma FFA concentrations is reduced in upper body obesity. High plasma FFA concentrations can induce a state of insulin resistance both in the muscle (glucose uptake) and in the liver (glucose release), independent of obesity. Thus, abnormal regulation of adipose tissue FFA export is a major component of the development of insulin resistance.

- Dysregulated production of a number of adipose-derived hormones, also called adipokines, is hypothesized to contribute to insulin resistance and the metabolic complications of obesity ***(LEE Goldman et al., 2012)***

- ***Islet Cell Failure and Type 2 Diabetes Mellitus***

Type 2 diabetes is generally the result of defects in both insulin secretion and insulin action. Many obese individuals are

insulin resistant, yet only a subset will develop diabetes mellitus. It follows that those who develop type 2 diabetes develop pancreatic β-cell decompensation with subsequent hyperglycemia. Animal (rodent) studies have suggested that a process referred to as lipotoxicity is involved in pancreatic β-cell failure. In this model, increased FFAs are proposed to contribute to the insulin secretory abnormalities seen in obesity and ultimately lead to β-cell failure. Another explanation for the development of β-cell failure in obesity is the overproduction of islet amyloid polypeptide. This protein is co-secreted with insulin and, because of its tertiary structure, can form toxicamyloid deposits in β cells. Amyloid deposits have been found in the pancreatic islets obtained at autopsy from patients with type 2 diabetes mellitus ***(LEE Goldman et al., 2012)***.

Respiratory System:

Fat accumulation on the thorax and abdomen decreases chest wall and lung compliance. Increased elastic resistance and decreased compliance of the chest wall further reduces total respiratory compliance while supine, leading to shallow and rapid breathing, increased work of breathing, and limited maximum ventilatory capacity. Respiratory muscle efficiency is below normal in obese individuals. Decreased pulmonary compliance leads to decreased functional residual capacity (FRC), vital capacity (VC), and total lung capacity (TLC).

Reduction in FRC is primarily a result of reduced expiratory reserve volume (ERV) but the relationship between FRC and closing capacity (CC), the volume at which small airways begin to close, is adversely affected (Fig.1). Residual volume and CC are unchanged. Reduced FRC can result in lung volumes below CC in the course of normal tidal ventilation, leading to small airway closure, ventilation-perfusion mismatch, right-to left shunting, and arterial hypoxemia. Forced expiratory volume in one second (FEV1) and forced vital capacity (FVC) are usually within normal limits. Obesity increases oxygen consumption and carbon dioxide production because of the metabolic activity of excess fat and the increased workload on supportive tissues. Obese patients retain their normal response to hypoxemia and hypercapnia. Chronic hypoxemia may lead to pulmonary hypertension and cor pulmonale ***(Barash et al., 2006)***.

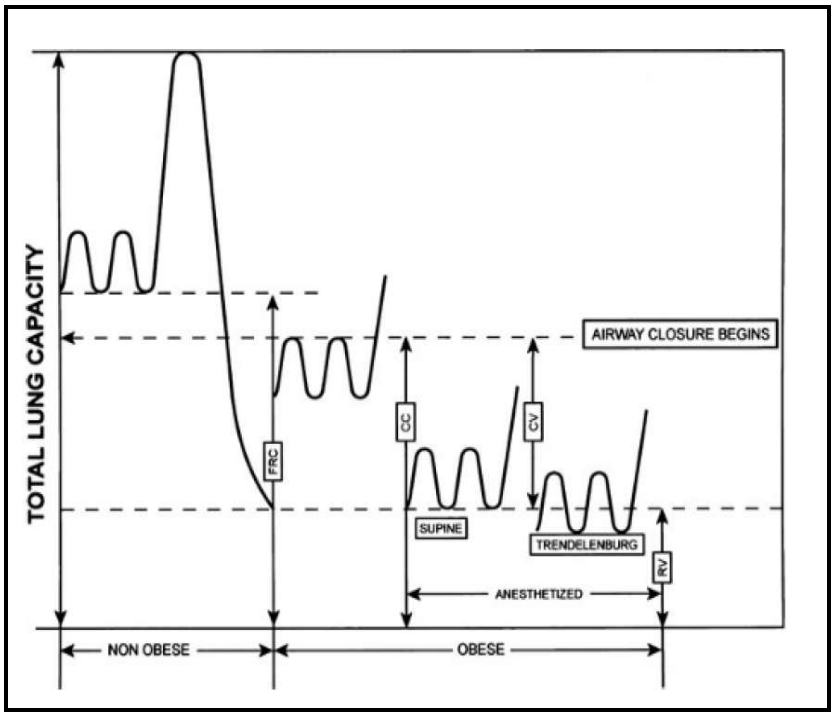

Fig. (1): Effects of obesity, positioning, and anesthesia on lung volumes. FRC, functional residual capacity; CC, closing capacity; CV, closing Barash PG, Cullen BF, Stoelting RK. Anesthesia and obesity. Clinical Anesthesia 2006; 2163:2183

Obstructive Sleep Apnea:

Up to 5% of obese patients have clinically significant *obstructive sleep apnea*. OSA is characterized by frequent episodes of apnea or hypopnea during sleep, snoring, and daytime symptoms, which include sleepiness, impaired concentration, memory problems, and morning headaches. Apnea is defined as 10 seconds or more of total cessation of airflow despite continuous respiratory effort against a closed glottis. A 50% reduction in airflow or a reduction sufficient