INTRODUCTION

Obesity is considered metabolic disease in which adipose tissue comprises a greater than normal proportion of body tissue and amount of fat tissue is increased beyond a point compatible with physical and mental health and normal life expectancy ***(Adams and Murphy, 2010)***.

Obesity is an epidemic that threatens the health of people all over the world. It is estimated that more than 1.7 billion people are overweight or obese. Decreased physical activity and over consumption of high-fat foods are the main contributing factors to the obesity epidemic. Conservative interventions to the treatment of obesity include early nutritional education, low-calorie diet, increased physical activity, and, sometimes, weight reducing medications. However, these treatments are not always effective, and many obese people undergo bariatric surgical procedures. There is a myriad of anesthesia challenges associated with these surgeries and other non-bariatric surgeries. A thorough understanding of the anesthetic considerations is essential to facilitate positive outcomes for these patients ***(Kuruba et al., 2007)***.

Understanding the pathophysiologic changes, surgical procedure, and the pharmacology of weight-reduction and anesthetic drugs is essential to provide high quality anesthetic care. The various comorbidities associated with obesity make the anesthetic management more difficult than usual.

Anesthetists must perform preoperative assessment to identify potential risk factors related to anesthesia and be adequately prepared for intraoperative challenges such as positioning, intubation, ventilation, and pain management. In addition, an obese patient is at higher risk for postoperative complications. Signs and symptoms of surgical complications may mimic medical complications making diagnosis difficult ***(Abeles et al., 2010)***.

AIM OF THE WORK

The goal of this essay is to focus the light on the recent updates in anesthetic management, postoperative complications and care of morbidly obese patients.

CHAPTER (1)

The World Health Organization (WHO) uses a class system to define obesity (Table 1). Statistics for 2013 from the UK, Health and Social Care Information Centre show that in adults, 24% of men and 25% of women are classified as obese and over 3% have class-3 obesity.

Table (1): Classification of obesity.

Body mass index; $kg.m^2$	Classification
< 18.5	Underweight
18.5–24.9	Normal
25.0–29.9	Overweight
30.0–34.9	Obese 1
35.0–39.9	Obese 2
> 40.0	Obese 3 (previously 'morbid obesity')

(Nightingale et al., 2015)

Etiology of obesity:

1- Genes:

The Fat mass and obesity-associated gene (FTO) was the first gene reliably associated with body mass index in genome-wide association studies on a population level. At present, the genetic variations within the FTO gene are still the common variants that have the largest influence on body mass index (***Bollepalli et al., 2010).***

The FTO gene is located on the long (q) arm of chromosome 16 and codes for a nuclear protein of the AlkB (Alkylation repair homologs) related non-haem iron superfamily, the exact physiological function of this gene is not know but studies in mice and humans indicate a role in nervous and cardiovascular systems and a strong association with body mass index, obesity risk, and type 2 diabetes (***Adeyemo et al., 2010).***

A cluster of single nucleotide polymorphism (SNPs) in the first intron of the *FTO* gene was found to correlate with an increase in body mass index (BMI) in both children and adults, regardless of gender it is unclear how FTO contributes to the etiology and increased susceptibility of obesity. Some reports suggest that it affects food intake, as carriers of the risk allele tend to choose high energy and palatable food ***(Wang et al., 2011).***

2-Leptin and obesity:

Leptin (from the Greek word "lepto'' meaning "thin") is a 167-amino acid peptide hormone secreted by white adipocytes. Blood leptin concentrations are increased in obese individuals. Leptin is a satiety hormone that provides negative feedback to the hypothalamus, controlling appetite and energy expenditure ***(Myers et al., 2010).***

Released into the circulation, leptin crosses the blood-brain barrier and binds to leptin receptors, influencing the

activity of various hypothalamic neurons, as well as encoding orexigenic and anorexigenic neuropeptides. Moreover, leptin affects a wide range of metabolic functions in the peripheral tissue ***(Vong et al., 2011).***

The physiological functions of leptin include reducing energy intake and increasing energy expenditure. Because these effects generally would result in weight loss, leptin resistance has been proposed as the mechanism by which humans with high leptin concentrations remain obese ***(Boustany et al., 2011)***

3-Physical activity:

A variety of studies conducted in children and youth show that physical activity is inversely related to excess body weight. Sedentary activities such as watching television, or playing videogames, etc, are conducive to becoming overweight ***(Hill et al., 2012).***

It's well known that physical activity had decreased recently and there has been a large shift towards less physically demanding work. The prevalence of labor saving technology is increasing at home and work (***Mozaffarian et al., 2011).***

4-Diet:

Dietary habits comprise an essential determinant for health, In the last few decades there has been a significant evidence that supports a series of associations between diverse dietary factors and chronic diseases, particularly cardiovascular

disease, cancer, diabetes, obesity. There are many dietary factors associated with high BMI such as: Dietary patterns of high energy density, frequent intake of sugared beverages, some evidence suggests a certain level of association between high ethanol intake and weight gain. The habitual intake of "fast food" (over once a week) might contribute to increased energy intake and to weight gain and obesity ***(Ruano et al., 2011)***.

Obesity is the result of a small and prolonged state of positive energy balance together with the decline in daily activity that came from industrialization, mechanized transportation, urbanization, and other aspects of technology ***(Estruch et al., 2013)***.

5-Sleeping patterns:

Recent epidemiological studies suggest that short sleep duration may be associated with the development of obesity from childhood to adulthood, and that sleep duration was also inversely associated with BMI and waist circumference (WC) after being controlled for potentially confounding variables. Intervention programs aiming for improving sleeping habits among child hood and adolescents need to consider such potential association of lifestyle variables with obesity ***(Di Milia et al., 2013).***

6-Gut microbiota and the development of obesity

The human gut harbors a highly diverse microbial ecosystem of approximately 400 different species, which is characterized by high inter-individual variability. The intestinal microbiota has recently been suggested to contribute to the development of obesity and the metabolic syndrome. Transplantation of gut microbiota from obese mice to non-obese, germ-free mice resulted in transfer of metabolic syndrome-associated features from the donor to the recipient. Proposed mechanisms for the role of gut microbiota include the provision of additional energy by the conversion of dietary fiber to short-chain fatty acids, effects on gut-hormone production, and increased intestinal permeability causing elevated systemic levels of lipopolysaccharides (LPS). This metabolic endotoxemia is suggested to contribute to low-grade inflammation, a characteristic trait of obesity and the metabolic syndrome. High-fat diet is discussed as another causal factor ***(Blaut and Klaus, 2012).***

A recently published review has shown the importance of studying how fatty acids, carbohydrates, micronutrients, and probiotics can influence gut microbiota composition and the management of obesity. Gut microbiota seems to be an important and promising target in the prevention and treatment of obesity and its related metabolic disturbances in future studies and in clinical practice ***(Boroni et al., 2012).***

7-Medical and psychiatric illness:

Certain physical and mental illnesses and the pharmaceutical substances used to treat them can increase risk of obesity. Medical illnesses that increase obesity risk include hypothyroidism, Cushing's syndrome, growth hormone excess, polycystic ovarian syndrome. Certain medications may cause weight gain or changes in body composition; these include insulin, sulfonylurea's, thiazolidinediones, atyprcal antipsychotics, antidepressants, steroids, certain anticonvulsants (phenytoin and valproate), pizotifen, and some forms of hormonal contraception ***(Boguszewski et al., 2010)***

8-Smoke cessation:

Increased BMI has also been associated with persons who have quit smoking. Studies demonstrated an average weight gain caused by smoking cessation of 4.4 kg in males and 5 kg in females who had stopped smoking within the last 10 years ***(Chiolero et al., 2008)***.

PATHOPHYSIOLOGY OF OBESITY

1-Effect of fat distribution:

Not all fat inside the body is identical. Unlike peripherally deposited fat, intra-abdominal fat is highly metabolically active and is known to be a contributor to several disease states. BMI alone is a poor predictor of co-morbidity, surgical, or anesthetic difficulty. Fat distribution is often more useful, waist or collar circumferences are more predictive of cardiorespiratory co-morbidity than BMI (***Ball and McAnulty, 2014)***.

Central obesity makes intra-abdominal surgery more difficult and is associated with increased fat deposition around the neck and airway (hence greater difficulty in airway management and ventilation of the lungs). Furthermore, the android fat distribution is associated with greater risk of metabolic and cardiovascular complications. Central obesity can be defined as a waist circumference greater than 88 cm in a woman and 102 cm in a man; or a waist-to-height ratio greater than 0.55 People who exhibit central, or visceral, obesity are often male and can be described as 'apple shaped', while those with a predominantly peripheral fat distribution are more likely to be female and are described as 'pear shaped' ***(Glance et al., 2010).***

The risk of cardiorespiratory and other co-morbidity increases with the duration of obesity ('fat years'). However, the presence and severity of co-morbidity may be masked by a sedentary lifestyle. The true significance of much obesity-related illness may only emerge during preoperative investigation or in the perioperative period (***Ball and McAnulty, 2014).***

2-Respiratory System:

Fat accumulation on the thorax and abdomen decreases chest wall and lung compliance. Reduced chest wall compliance results in part from the weight of adipose tissue around the thoracic cage and increased pulmonary blood volume because of an overall increase in blood volume as more volume is required to perfuse the additional body fat. Decrease lung compliance, cephalic displacement of abdominal contents, and increased thoracic blood volume contribute to reduced functional residual capacity (FRC). FRC declines steeply with increasing BMI and reaches values of around 1 liter or less in subjects whose BMI exceeds 40 ***(Ball and McAnulty, 2014).***

Reduced FRC can result in lung volumes below closing capacity (CC : the volume at which small airways begin to close) in the course of normal tidal ventilation leading to small airway closure, ventilation perfusion mismatch, right-to left shunting, and arterial hypoxemia ***(Schumann, 2013).***

Decrease lung compliance may be accentuated during laparoscopic surgery, particularly if the excessive pneumoperitoneal insufflations pressures are used. Although some authors have reported an improvement in respiratory mechanics when the reverse Trendelenburg position is used, this has not been a universal finding, and may be a further consequence of diaphragmatic splinting ***(Lemanu et al., 2012).***

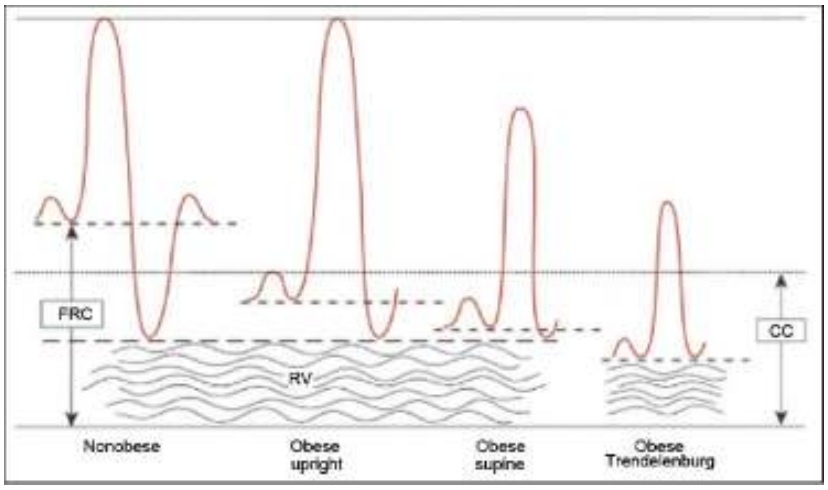

Figure (1): Changes in TV, FRC, in obese patients with different positions ***(Lemanu et al., 2012).***

Airway resistance is increased and correlates with BMI, it is further increased when transferring from a sitting to supine position. These factors contribute to an increased work of breathing and increase oxygen consumption. The combined effect of these changes is a tendency to hypoxemia at rest which is accentuated in the supine position ***(Schumann, 2013).***

General anesthesia accentuates these changes. A 50% decrease in FRC occurs in obese anesthetized patients compared with a 20% decrease in non-obese individuals. The decrease in FRC impairs the ability of obese patients to tolerate periods of apnea, such as during direct laryngoscopy for endotracheal intubation. Obese individuals are likely to experience oxygen desaturation following induction of anesthesia despite adequate preoxygenation. This phenomenon reflects a decreased oxygen reserve secondary to the reduced FRC and an increase in oxygen consumption resulting from the increased metabolic activity of excess adipose tissue ***(Schumann, 2013).***

3- Sleep-Disordered Breathing:

Sleep-disordered breathing (SDB) describes the spectrum of conditions ranging from obstructive sleep apnea (OSA) to obesity hypoventilation syndrome (OHS). Each of these conditions has a spectrum of severity, described according to the number and severity of oxygen desaturation occurring every hour and their impact upon the patient ***(Berry et al., 2012).***

A) Obstructive sleep apnea:

OSA is defined as apnea episodes secondary to pharyngeal collapse that occur during sleep, it may be obstructive, central, or mixed. The incidence of OSA increases with obesity and increasing age. More than 95% of cases go

unrecognized. The diagnosis is confirmed by sleep studies. The characteristic features are:

(i) Frequent episodes of apnea or hypopnoea during sleep, where five or more per hour or 30 per night are often quoted as clinically significant. An apnea episode is defined as 10 seconds or more of total cessation of airflow, despite continuous respiratory effort against a closed airway. Hypopnea is a reduction in the size or number of breaths compared with normal ventilation and is associated with some degree of arterial desaturation of 4% or more.

(ii) Snoring.

(iii) Day-time somnolence, associated with impaired concentration and morning headaches.

(iv) Pathophysiological changes: hypoxemia (leading to secondary polycythaemia), hypercapnia, systemic vasoconstriction, or pulmonary vasoconstriction leading to right ventricular failure.

Patients with OSA frequently have increased adipose tissue in the pharyngeal wall, particularly between medial and lateral pterygoids. This results in increased pharyngeal wall compliance, with a tendency to airway collapse when exposed to negative pressure resulting in turbulent airflow and snoring. In susceptible individuals this may progress to severe snoring and, ultimately, to sleep apnea. Sleep fragmentation is the most

likely explanation for the daytime somnolence, which is associated with impaired concentration, memory problems, In addition, patients may complain of morning headaches caused by nocturnal carbon dioxide retention and cerebral vasodilatation (***Mutter et al., 2014).***

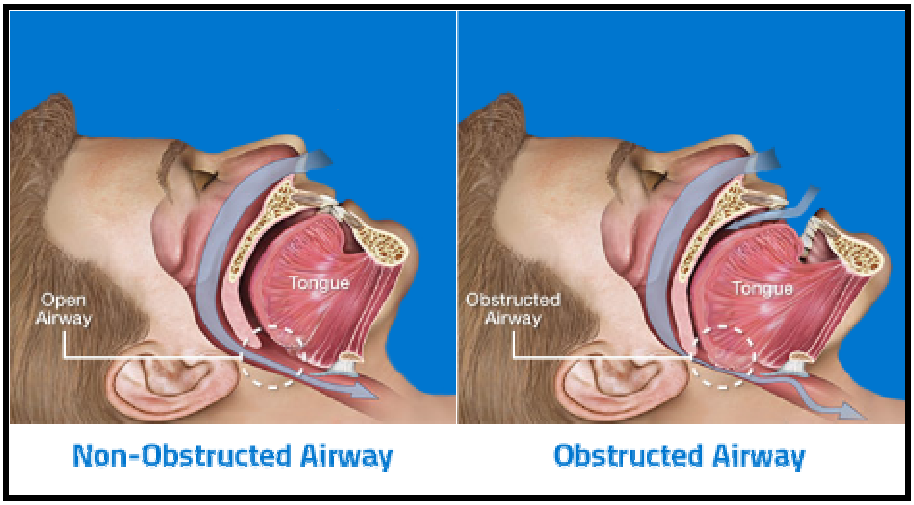

Figure (2): Mechanism of OSA ***(Mutter et al., 2014).***

Severe OSA occurs in 10–20% of patients with BMI > 35 and is often undiagnosed. The overall diagnosis of OSA is associated with a greater than doubling of the incidence of postoperative desaturation, respiratory failure, postoperative cardiac events and ICU admission. The presence of multiple and prolonged oxygen desaturations increases the sensitivity to opioid induced respiratory depression However, if identified pre-operatively and treated appropriately with continuous positive airway pressure (CPAP), the risk of complications is much reduced ***(Mutter et al., 2014).***