

INTRODUCTION

Obesity is a medical condition in which excess body fat has accumulated to that extent that it may have a negative effect on health leading to reduced life expectancy and increased health problems. Obesity is becoming increasingly prevalent in developed countries and anesthesiologists will face larger numbers of obese patients undergoing bariatric surgery. The prevalence of obesity doubled in America between 1976-1980 and 1999-2000 increasing from 15.1 percent to 30.9 percent. Obesity is the result of genetic, behavioral, environmental, physiological, social, and cultural factors that result in energy imbalance and promote excessive fat deposition (*Mathier and Ramanathan, 2007*).

In obese patients, the levels of catecholamines and cortisol are often increased and contribute to metabolic and vascular abnormalities. Obesity is associated with important co-morbidities (e.g, diabetes mellitus, arterial and pulmonary hypertension, obstructive sleep apnea syndrome) which increase the risk of peri-operative complications (*Grassi et al., 2009*).

Patients should be assessed by a multidisciplinary team, which may include endocrinologists, dieticians, psychologists, specialist nurses, and experienced surgeons and anesthesiologists. The pre-operative assessment of the patient is crucial in identifying and stratifying risk to ascertain level of

perioperative care required, and also each individual's suitability for surgery (*Brodsky et al., 2002*).

There are two types of bariatric surgeries; restrictive surgery and malabsorption surgery. Morbidly obese patients require special care in positioning during operation and certain anesthetic considerations are often to be considered (*Barak et al., 2014*).

Morbidly obese patients are more likely to be immobile postoperatively and are at risk of deep vein thrombosis and pulmonary embolism, with a combined incidence of approximately 2%. Optimal analgesia ensures adequate ventilation and good pulmonary mechanics which reduce the risk of postoperative chest infections. It has been shown that most laparoscopic bariatric patients have little pain with adequate local anesthetic wound infiltration and a patient-controlled analgesia (*McNatt et al., 2007*).

DEFINITION, EPIDEMIOLOGY AND ETIOLOGY OF OBESITY

Obesity is **defined** as abnormal or excessive fat accumulation that may impair health. Body mass index (BMI) is a simple index of weight-for-height that is commonly used to classify overweight and obesity in adults. It is defined as a person's weight in kilograms divided by the square of his height in meters (kg/m^2) (**Table 1**).

The World Health Organization (WHO) **definition** is that BMI greater than or equal to 30 is obesity. BMI provides the most useful population-level measure of obesity as it is the same for both sexes and for all ages of adults. However, it should be considered a rough guide because it may not correspond to the same degree of fatness in different individuals (*WHO Media Center, 2015*).

Prevalence of obesity according to WHO:

Classification of obesity

Overweight and obesity are classified according to BMI, waist circumference, and associated disease risk (**Table 1**).

Table (1): Classification of overweight and obesity by BMI, waist circumference, and associated disease risk

	BMI (kg/m ²)	Obesity class	Disease risk (relative to normal weight and waist circumference)	
			Men ≤ 40 in (102 cm)	>40 in (102 cm)
			Women ≤ 35 in (88 cm)	>35 in (88 cm)
Underweight	<18.5		-----	-----
Normal	18.5 to 24.9		-----	-----
Overweight	25.0 to 29.9		Increased	High
Obesity	30.0 to 34.9	I	High	Very high
Morbid Obesity	35.0 to 39.9	II	Very high	Very high
Extreme obesity	≥40	III	Extremely high	Extremely high

(National Institutes of Health, National Heart, Lung, and Blood Institute, 1998)

In 2013, 42 million children under the age of 5 were overweight or obese. Once considered a high-income country problem, overweight and obesity are more recently on the rise in low- and middle-income countries, particularly in urban settings. In developing countries with emerging economies (classified by the World Bank as lower- and middle-income countries), the rate of increase of childhood overweight and obesity has been more than 30% higher than that of developed countries (*WHO Media Center, 2015*).

In 2014, more than 1.9 billion adults, 18 years and older, were overweight. Of these over 600 million were obese. Overall, about 13% of the world's adult population (11% of men and 15% of women) were obese in 2014. In 2014, 39% of adults aged 18

years and over (38% of men and 40% of women) were overweight. The worldwide prevalence of obesity more than doubled between 1980 and 2014 (*WHO Media Center, 2015*).

Etiology of Obesity

Although obesity is most commonly caused by excess energy consumption (dietary intake) relative to energy expenditure (energy loss via metabolic and physical activity), the etiology of obesity is highly complex. It includes genetic, psychological, environmental, social, economic and even political factors that interact in varying degree to promote the development of obesity (*Aronne et al., 2009*).

Contributing Factors to the Obesity Epidemic:

1. Food environment.
2. Decreases in physical activity.
3. Drug– induced weight gain.
4. Sleep debt.
5. Decline in cigarette smoking.
6. Endocrine disruptors.
7. Increasing gravid age and intrauterine effects.
8. Greater reproductive fitness of higher BMI individuals yielding the selection of obesity- predisposing genotypes
9. Changes in policy.
10. Infections.

1. Food Environment

The food, or "built" environment has shifted in ways that promote over eating: highly caloric and fat-laden foods are not only affordable, but also easily accessible (i.e., numerous fast food restaurants, vending machines of energy dense items in schools and offices). These highly palatable foods are frequently available in large portions which contribute to increased daily caloric intake. The majority of products in grocery stores are high caloric and frequently consumed by millions of families (*Rolls, 2003*).

2. Decreases in Physical Activity

Physical activity levels have also dramatically decreased in the past several decades. It has been estimated that less than half of US adults are engaged in recommended levels of physical activity in 2005. Levels of physical activity have also decreased in adolescents. There are less access to physical activity places (fewer sidewalks) and less physical education in schools. The myriad advances in technology developed over the past few decades have made many tasks more efficient, but in the process have ultimately decreased the number of calories expended (i.e., TV remote controls, automatic garage door opener, etc.) (*Centers for Disease Control, 2013*).

3. Drug- Induced Weight Gain

Weight gain is associated with several commonly used medications including psychotropic medications, diabetic treatments, anti hypertensives, steroid hormones and contraceptives, antihistamines, and protease inhibitors. The deleterious effects of drug-induced weight gain include, paradoxically, increased risks for developing type 2 diabetes, hypertension, hyperlipidemia, as well as poor medication compliance (*Aronne and Segal, 2003*).

4. Sleep Debt

Sleep debt has also been linked to increased body weight. Studies have shown that hours of sleep per night are negatively correlated with BMI. Sleep restriction has been showed to increase the hunger and appetite (*Gangwisch et al., 2005*).

5. Decline in Cigarette Smoking

The relative decline in cigaratte smoking may also be a factor that has contributed to the obesity epidemic. Studies have shown that weight gain is a common sequel of smoking cessation. Furthermore, smokers typically weigh less than non smokers (*Filozof et al., 2004*).

6. Endocrine Disruptors

Endocrine disruptors which are industrially –produced substances that can affect endocrine function, may be a contributing factor to the etiology of obesity. They include

dichlorodiphenyltrichloroethane, some polychlorinated biphenols and some alkylphenols, that may act by disrupting endogenous hormonal regulation (*Keith et al., 2006*).

7. Increasing Gravid Age and Intrauterine Effects

Increasing gravid age, intrauterine effect and greater reproductive fitness of higher BMI individuals yield the selection of obesity-predisposing genotypes (*Keith et al., 2006*).

8. Obesity Predisposing Genotypes

Although the genetics of obesity is a highly researched area, just a small number of rare single genetic abnormalities have been discovered. Current research on the epigenetics of obesity is investigating the influence of behavioral and environmental factors on genetic expression (*Swinburn et al., 2011*).

9. Changes in Policy

For instance, United States of America (USA) farm subsidy policies may have caused certain foods to be more abundant and cheaper, potentially contributing to lower relative prices and increased consumption of fattening food (*Wallinga, 2010*).

10. Infections

Infections may also play a role in the etiology of obesity. Adenovirus-36 infections has been shown to cause obesity in animals. Studies have shown that obese individuals are more likely to have been infected with Adenovirus-36 (*Pasarica et al., 2006*).

PATHOPHYSIOLOGY OF OBESITY

Obesity is an exaggeration of normal adiposity and is a central player in the pathophysiology of diabetes mellitus, insulin resistance, dyslipidemia, hypertension, and atherosclerosis, largely due to its secretion of excessive adipokines. Obesity is a major contributor to the metabolic dysfunction involving lipid and glucose, but on a broader scale, it influences organ dysfunction involving cardiac, liver, intestinal, pulmonary, endocrine, and reproductive functions.

Disorders Associated with Obesity

1. Respiratory Diseases
2. Insulin Resistance and Type 2 Diabetes
3. Atherogenic Dyslipidemia
4. Metabolic Syndrome
5. Hypertension and Other Cardiac Diseases
6. Cancers
7. Liver and Gallbladder Disease
8. Kidney Disease
9. Gastrointestinal Disease
10. Osteoarthritis
11. Effect of Obesity on Fertility
12. Other Endocrinal Effects of Obesity
13. All-Cause Mortality

1. Respiratory Diseases

A. Obstructive Sleep Apnea (OSA)

OSA is defined as apneic episodes secondary to pharyngeal collapse that occur during sleep; it may be obstructive, central, or mixed. Individuals with sleep apnea are often unaware of their sleep disorder. It is usually first recognized as a problem by family members who witness the apneic episodes or is suspected by their primary care doctor because of the individual's risk factors and symptoms. Individuals with untreated OSA can stop breathing hundreds of times a night during their sleep. These apneic events can lead to fragmented sleep that is of poor quality, as the brain arouses briefly in order for the body to resume breathing. Untreated sleep apnea can have direct health consequences and can increase the risk of hypertension, diabetes, heart disease, and heart failure (*Tishler et al., 2003*).

Epidemiology

OSA affects more than twelve million Americans and it is estimated to be as prevalent as asthma and diabetes. An epidemiological review estimates that 1 in 5 adults has at least mild OSA and 1 in 15 adults has at least moderate OSA. Given the fact that obesity is a major risk factor for OSA, and given the global rise in obesity, the prevalence of OSA will increase in the future. Sleep apnea can affect anyone at any age, even children. Although OSA is becoming increasingly prevalent;

however, because of the lack of awareness by the public and health-care professionals, the vast majority remain undiagnosed and untreated (*Young et al., 2002*).

Primary risk factors for sleep apnea (Tishler et al., 2003)

1. Weight gain or being overweight with a BMI >30 Kg/m².
2. Neck circumference [17 inch (or 43.2 cm) in men; 16 inch or (40.6 cm) in women].
3. Age >40.
4. Male gender.
5. Structural factors related to craniofacial anatomy.
6. Ethnicity.
7. Family history of sleep apnea.

Pathophysiology

The International Classification of Sleep Disorders, (ICSD) was published by the American Academy of Sleep Medicine to standardize definitions and create a systematic approach to the diagnosis of sleep disorders. The ICSD subdivides sleep disorders into eight major categories, one of which is sleep-disorder breathing (SDB) (*Thorpy, 2012*).

The ICSD further classifies SDB into three basic categories: Central sleep apnea (CSA) syndromes, OSA syndromes, and sleep-related hypoventilation/ hypoxic syndromes. OSA involves a complete cessation or a significant

decrease in airflow in the presence of breathing effort. CSA is the cessation of airflow with an absence of breathing effort. Breathing effort is measured by abdominal and/or chest movement (*White, 2005*).

The human airway is composed of soft tissue that can collapse during rapid eye movement (REM) sleep, when the muscle tone of the body relaxes. Larger soft tissue mass or abnormal tissue deposits can also increase extraluminal tissue pressure and lower the threshold for airway collapse. In normal, non obese individuals without OSA, muscle relaxation during sleep does not completely collapse the airway. However, airway collapse can occur during muscle relaxation when there is a pathological increase in tissue pressure, as a product of extra soft tissue mass (in a normal-sized enclosure) and/or structural limitations (small maxillary or mandibular compartment) with normal tissue mass. Chronically, this dysfunction can cause problems with the regulation of pharyngeal dilator muscle activation (which plays an important role in maintaining airway patency) in patients with OSA (**Figure 1**) (*White, 2005*).

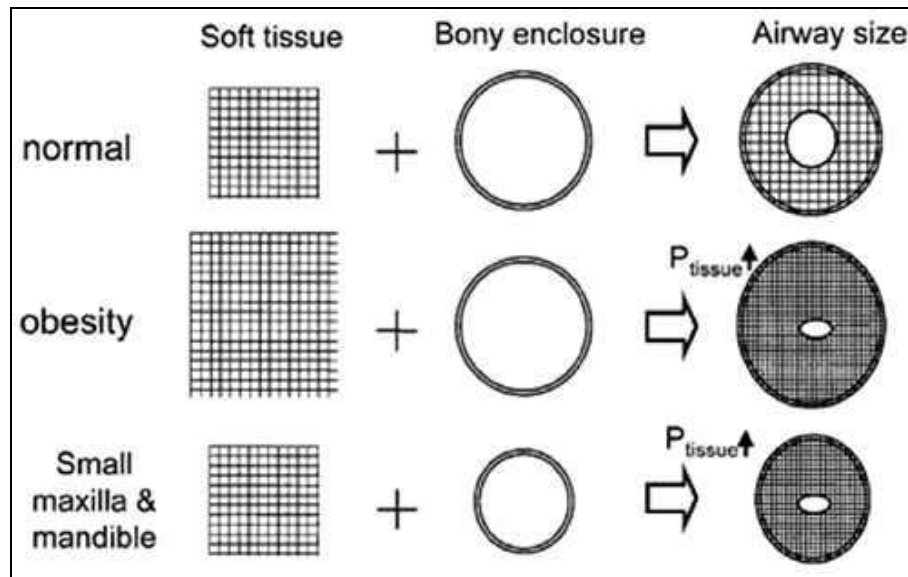


Figure (1): The difference of soft tissue, bony enclosure and airway size in normal, obesity and small maxilla & mandible (*White, 2005*).

P_{tissue}: Tissue pressure

OSA is thus characterized by the partial or total collapse of the pharyngeal airway during sleep and the need to arouse to resume ventilation. In adults, the obstruction typically occurs at the level of the oropharynx (*Eckert and Malhotra, 2008*).

Chronic severe OSA can result in prolonged hypoxemia, sleep deprivation, and other complications. With most apneic events, the brain briefly arouses in order for the body to resume breathing, but consequently, sleep is extremely fragmented and of poor quality (*Eckert and Malhotra, 2008*).

Several additional anatomical factors play a role in OSA. These include the position that the patient sleeps in, airway reactivity and airway secretions. Position can have a strong influence on airway patency. Because the airway is collapsible,

gravitational forces can cause the retropulsion of the tongue and soft palate while laying supine, thus generating increased positive tissue pressure and narrowing the airway. For this reason, OSA worsens in the supine sleeping position for most individuals (*Eckert and Malhotra, 2008*).

Diagnosis

The American Academy of Sleep Medicine (AASM) defines an apnea as a cessation in airflow lasting at least 10 seconds; apneic episodes can last anywhere from 10 seconds to one minute, and may occur multiple times per hour. Hypopnea is defined as a recognizable transient reduction (but not complete cessation) of breathing for at least 10 seconds. This differs from apnea in that there remains some flow of air. In the context of sleep disorders, a hypopnea event is only considered to be clinically significant if there is a 30% or more reduction in air flow with an associated 4% or greater desaturation in O₂ level, lasting for 10 seconds or longer, or if it is associated with an arousal or fragmentation of sleep. Apneas and hypopneas are both considered in assessing the severity of a person's sleep disorder. (*Thorpy, 2012*).

The Apnea-Hypopnea Index (AHI) is an index used to indicate the severity of sleep apnea. It is represented by the number of apnea and hypopnea events per hour of sleep. The apneas must last for at least 10 seconds and be associated with a decrease in blood oxygenation. In general, an individual is

considered to have an OSA syndrome if they demonstrate an AHI of at least 5 with the presence of daytime symptoms (excessive daytime sleepiness, loud snoring, morning headache, waking up with a sore throat or dry mouth and attention problems) or AHI of 15 or more independent of symptoms. The AHI can be also used to stratify the severity of the disease; an AHI of 5–15 is classified as mild, 15–30 is considered moderate and greater than 30 is considered severe (*Eckert and Malhotra, 2008*).

Another measure that is often used is the Respiratory Disturbance Index (RDI). Like the AHI, RDI measures respiratory events; however, it also includes respiratory event-related arousals (RERAs). RERAs are arousals from sleep that do not technically meet the definitions of apneas or hypopneas, but do disrupt sleep. Some research studies have found that 30% of symptomatic patients would have been left untreated if the AHI were used rather the RDI (*Eckert and Malhotra, 2008*).

B. Pickwickian {Syndrome Obesity hypoventilation syndrome (OHS) }

Pickwickian syndrome is a complex of symptoms that primarily affect patients with extreme obesity. The major health problem that occurs in patients with this disease is sleep apnea. This is caused in part by the excess amounts of fatty tissue surrounding the chest muscles. This excess fat places a strain on the heart, lungs, and diaphragm of the patient, making it