Introduction

Obesity has become major health problem in both developed and developing countries, in twenty first century. Because of it's high prevalence and causal relation ship with serous medical and psychological complications ***(Compbell and Haslam, 2005).***

Obesity is excessive amount of body fat due to increase number and size of fat cells which frequently result in significant impairment of health. While ***over weight*** is excessive amount of body structures including fat, muscles, bone and water. In some people such as athletes with lot of muscle can be over weight without being obese ***(Anne Collins, 2006).***

Obesity is the most common form of malnutrition in which little is known about the aetiology of obesity. There are propably spectrum of different kinds of disorders as genetic, behaviore or both which increase intake or decrease expenditure of an obese individual ***(Jensen, 2004).***

Physical examination including measurement of weight and height is usually sufficient to diagnose obesity. But currently, body mass index (BMI) is preferred formula it's useful indicator to mesure obesity and man correlates with body fat and metabolic complications of obesity. According to BMI obesity defines as 30 Kg/m^2 over desirable weight where as BMI over 40 Kg/m^2 defines sever obesity. More recently, another category, super obesity has been defined as BMI greater than 50 $Kg\backslash m^2$ ***(Bray, 2003).***

The risk of metabolic complications and co morbid factors is related to both BMI especially greater than 25 Kg/m^2 and waist circumference ***(C. Summertan et al., 2004).***

Reduction body weight can be achieved through medical treatment and/or surgical treatment.

Medical treatment including therapeutic management and conservative management which including behavioral modification, dietary modification, exercise programs and appetite suppressant. All of these measures have not been effective in patient with marked obesity ***(American Society for Bariatric Surgery, 2005).***

The problem in medical treatment of morbid obesity is not only to lose weight but also to maintain the weight loss and estremely high relapse after 1-2 years ***(Desai and Eugon, 2002)***.

Surgical treatment is only proven method to achieve long term weight contral for morbid obesity about 30% ***(Sugerman, 2001)***.

Table (1): Gastrointestinal surgically designed approaches to weight loss

Approach	Operations
-Global mal-absorption	*-Jejuno-ileal bypass or small bowel bypass*
-Gastric restriction	*-Gastroplasty. & -Gastric stapling* *-Vertical banded gastroplasty* *-Gastric banding*
-Combined gastric restriction and bypass.	*-Roux-en-Y gastric bypass*
-Selective mal-digestion and mal-absorption with partial gastric restriction with gastrectomy.	*-Partial bilio-pancreatic bypass with Gastrectomy or duodenal switch* *-Distal gastric bypass*

Gastric bypass is one of surgical operation: that treat morbid obesity, it was first recommended by Mason and Ito 1967 in this procedure stomach in the region of gastric fundus is separated from remaining portion of the stomach and small fundus pouch is anastomased with down up loop of jejunum ***(Masson and Ito, 1969).***

Aim of the study

The aim of this study is discussion of different surgical procedures especially role of gastric bypass in management of morbid obesity and its advantages, disadvantages and complications in the field of bariatric surgery especially after increase the interest and acceptance of surgical management as golden role in treatment of morbidly obese patients.

Stomach Anatomy

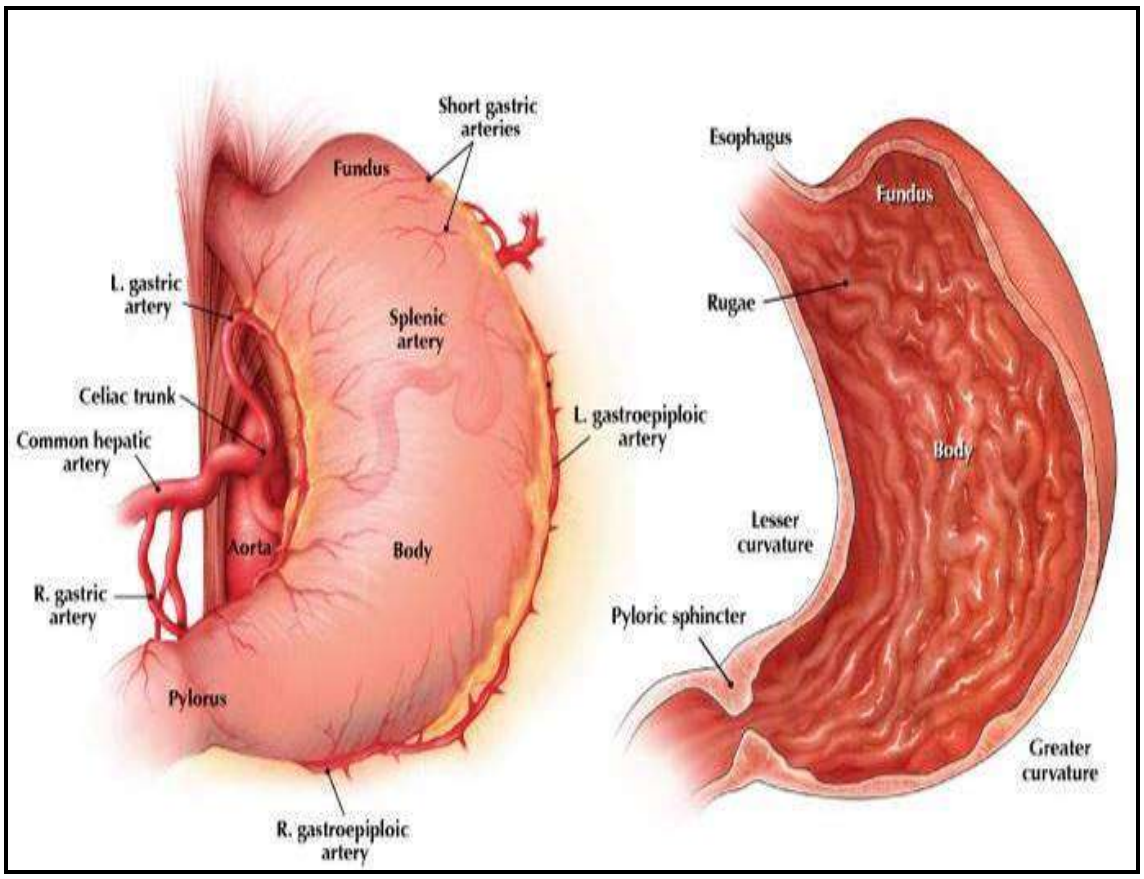

Fig. (1): Anatomy of stomach

The stomach is roughly J-shaped structure occupies the epigastrium and left hypochondrium regions. ***(Snell, 2004)***

***** Both shape and size of the stomach vary greatly as follows:***

It tends to be high and transverse in the "short" obese persons, and "elongated" in thin persons, even in the same person, the shape depends on three factors; fullness, body position and phase of respiration ***(Snell, 2004).***

** The **stomach is divided into four parts;**

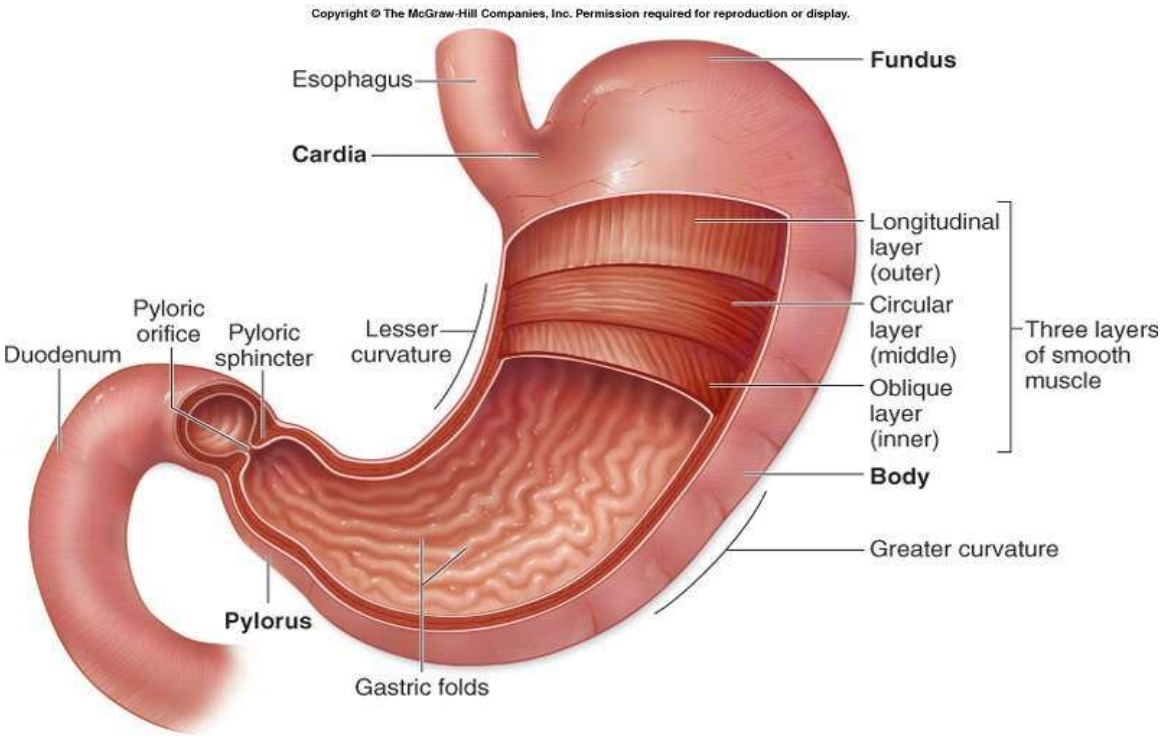

Fig. (2): Parts of stomach:

The cardiac orifice** is rather fixed and lies in the upper part of the epigastrium in contact with the left lobe of the liver ***(Keith moor and Arthur Dalley, 1999).

****Pyloric part** itself is divided in to antrum (Proximal dilated part), canal (distal narrow tail) and sphincter (which surrounds the pyloric orifice). ***fundus** is the dome-shaped part of the stomach, which projects to the left and above a horizontal line from the cardiac orifice to the greater curvature ***(Neil Borley, 2005)***.

****The lesser curvature:** it forms the right border of the stomach; it extends from the right side of the cardiac orifice to the upper border of the pylorus. The lowest part of the lesser curvature is

represented by a notch called incisura angularis. The right and left gastric vessels run along lesser curvature. ***(Neil Borley, 2005)***.

****Greater curvature;** It forms the convex left and lower border of the stomach, at the left and upper part of the greater curvature the fundus of the stomach is connected to the spleen by the gastrosplenic ligament, while from the rest of the greater curvature the greater omentum descends to reach the transverse colon ***(Neil Borley, 2005)***.

***Body of the stomach** is a region between the fundus and incisura angularis on the lower part of lesser curvature to the greater curvature.

****Surfaces and peritoneal coverings:***

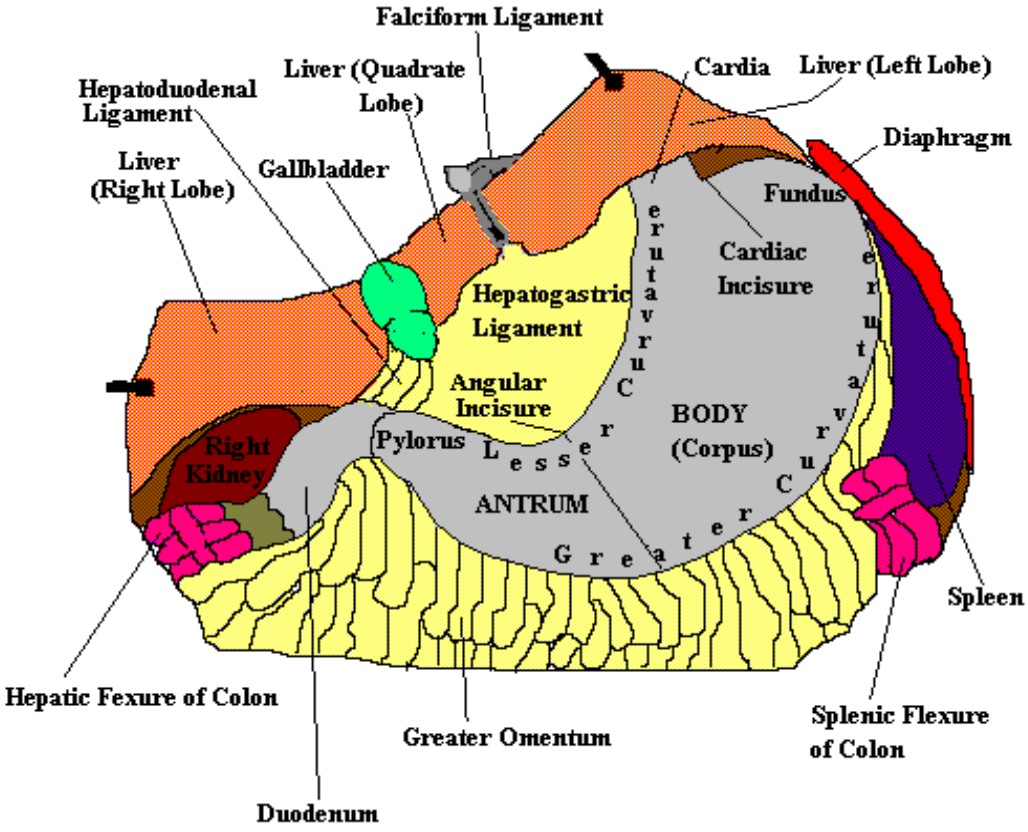

Fig. (3): Surfaces and peritoneal coverings:

The stomach has two surfaces; anterior and posterior, which are covered by peritoneum except a small triangular area in the posterior surface near the cardiac, and where the stomach is directly related to the left crus of the diaphragm which is called **"bare area of the stomach"** ***(Snell, 2004)***.

The stomach is related to the greater sac of peritoneum in front and to the lesser sac behind ***(Snell, 2004).***

Relations of the stomach:

**** Antero-superior surface is subdivided to:***

Fundus,** related to heart and pericardium. Left lobe of the liver is at direct contact with the anterior surface over a narrow area adjoining the lesser curvature ***(Neil Borley, 2005).

****The lower and right part** of the anterior surface lies at direct contact with the anterior abdominal wall in the region of the upper part of the left rectus muscle and linea Alba ***(Neil Borley, 2005)***.

*****The upper and left part** of the anterior surface lies under cover of the left costal margin and is in contact with left half of the diaphragm, which separates this part of the stomach from the left pleura and base of the left lung.

*****Relations of the Postero-inferior surface:***

Which forms a large part of the anterior wall of the lesser sac of the peritoneum. The cavity of the lesser sac separates the posterior surface of the stomach from many structures, which together form what is usually known as the stomach bed. There are 8 structures arranged in this order from below upwards; transverse colon,

transverse mesocolon, body of pancreas, Splenic artery, upper part of the left kidney, left suprarenal gland, Spleen and left crus of diaphragrn ***(Neil Borley, 2005).***

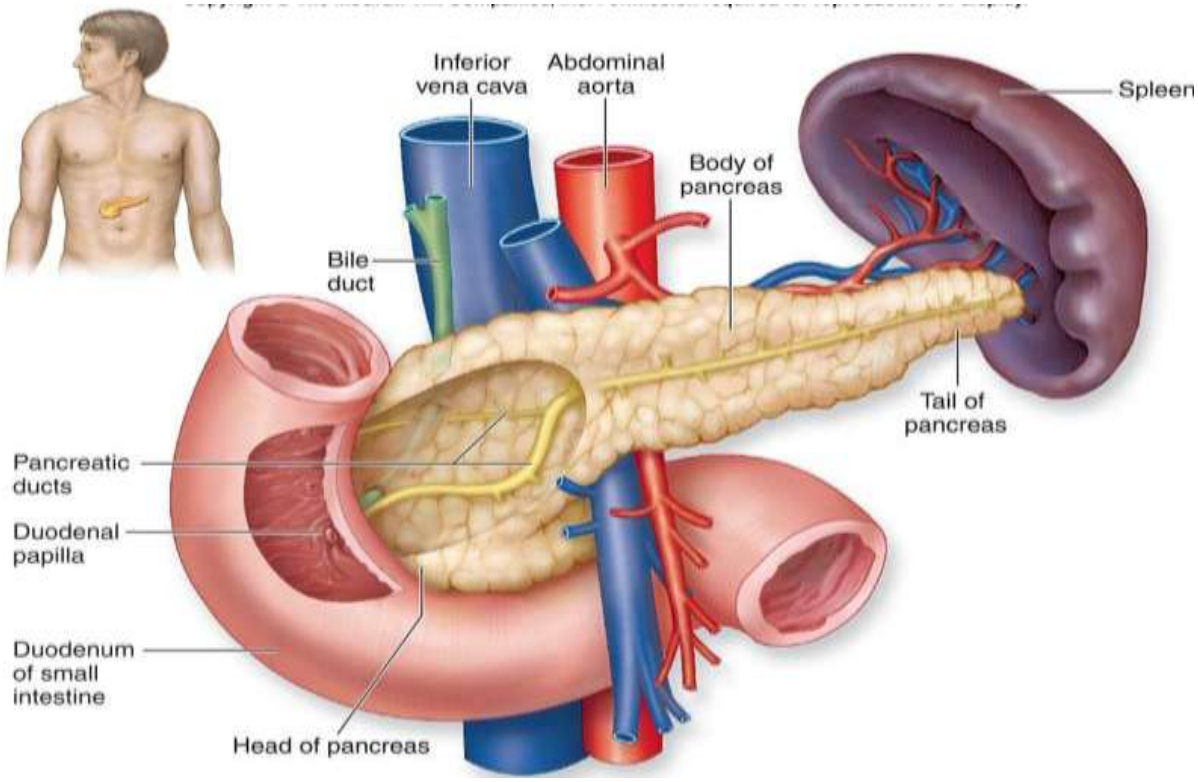

Fig. (4): Relations of the Postero-inferior surface (stomach bed)**:**

Blood Supply of the stomach*:*

****Arterial blood -supply***

The stomach is richly supplied with arteries either from the celiac trunk itself, or from its branches. Five arteries supply the stomach; **left and right gastric artery**, **right and left gastroepiploic arteries and short gastric arteries** ***(Neil Borley, 2005)***.

Venous drainage:

Veins, which correspond to the arteries, drain into the portal circulation. **Right and left gastric veins** drain into the portal vein itself. **The left gastroepiploic and short gastric veins** join the splenic vein**. The right gastroepiploic** vein drains into the superior mesenteric vein ***(Neil Borley, 2005)***.

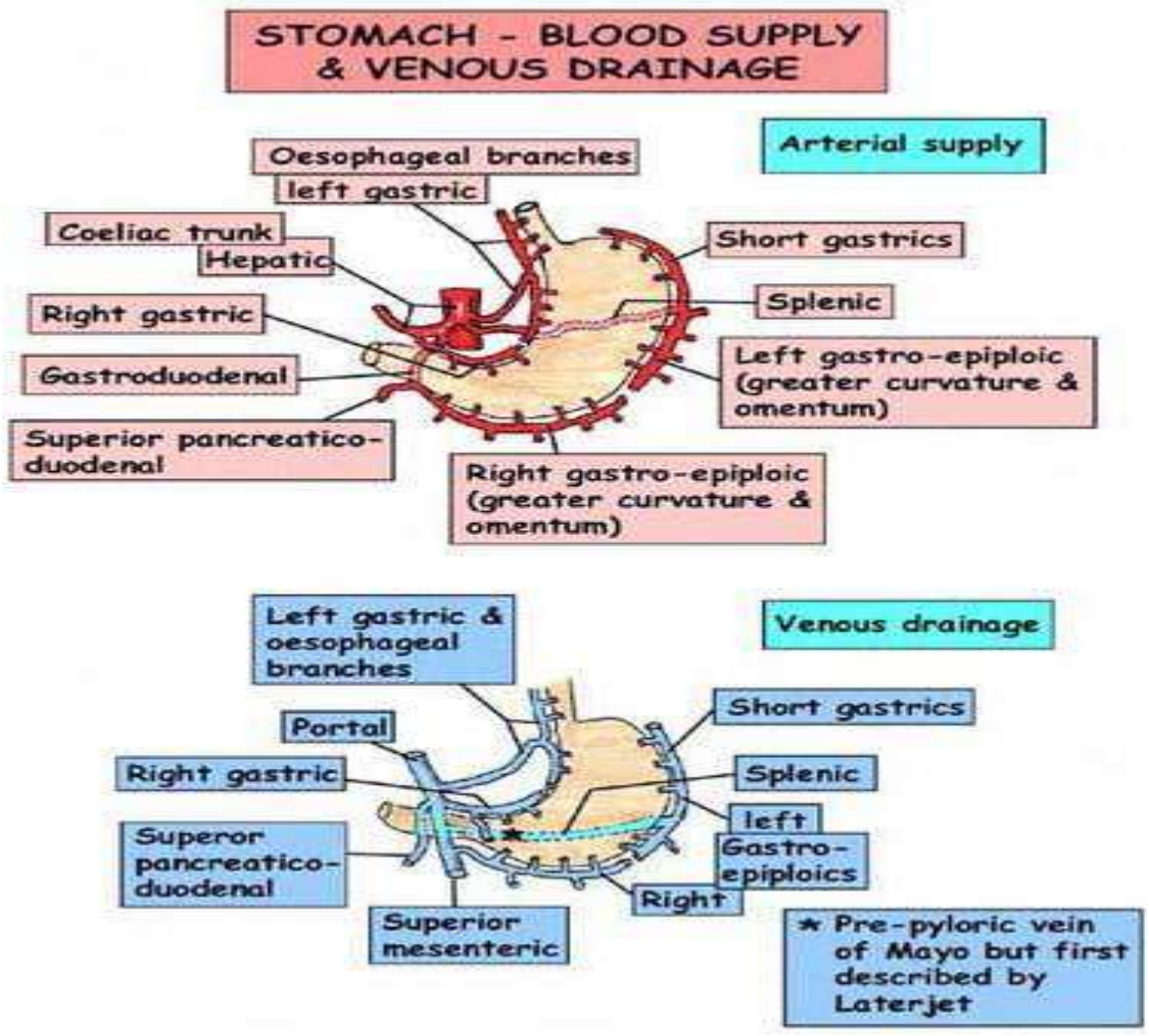

Fig. (5): Blood supply of stomach

******Lymph drainage:***

The abundant lymph vessels of the stomach originate in the mucosa and ramify in plexus of the submusculosa. These channels pour into lymph nodes; mainly along the **greater and lesser curvature group *(Snell, 2004).***

The second group includes ***hepatic and sub pyloric glands***. The third group is in continuity with the lymph nodes along the **greater curvature of the stomach and drains its lower 2/3 *(Snell, 2004).***

**** Nerve Supply:***

Sympathetic innervations: emerge from **T5, 6, 7 and 8** through the **celiac plexus** by its extensive distribution around the gastric and gastroepiploic arteries.

** ***Parasympathetic innervations:*** provided via the **vagus nerve trunk**, this nerve trunk entail **anterior and posterior vagi**.

*** I) Anterior vagus Trunk:** It gives off; **hepatic, gastric branches** & **anterior long nerve of the antrum**.

**** II) Posterior vagus Trunk:** Divided in to **celiac, gastric and posterior long nerves of the antrum *(Snell, 2004).***

Physiology

Physiology of feeding & Regulation of food intake

1- Glucose utilization by the satiety center:

There is a considerable debate about the signals that are sent by the satiety and feeding centers, which are found in the Ventro medial nucleus (VMN) and paraventricular nucleus (PVN) of the hypothalamus to regulate food intake. The activity of the satiety center is probably governed in part by the level of glucose utilization of cells within the center ***(Bray, 2002).***

2- The Limbic System:

The limbic system is also involved in the neural regulation of appetite. The ascending noradrenergic fibers in the ventral bundle inhibit appetite ***(Mills, 2002).***

3- Effects of neurotransmitter on brain centers that control feeding:

The catecholamine and beta-adrenergic stimulation inhibits eating behavior. Nor epinephrine, Serotonin, Histamine and a number of peptides may be involved in transmission of information that regulates food intake and nutrient stores ***(Bray, 2003).***

4- Degree of distention of the stomach:

There are stretch which is probably situated in the smooth muscles of the stomach. They send afferent impulses through vagus

nerve fibers to the hypothalamic satiety centers and they are stimulated by the mechanical distension of the stomach ***(Campbell and Haslam, 2005)***

5- Small Intestine:

There are several mechanisms by which the small bowel is thought to mediate satiety. There are osmo-receptors in the duodenum that may cause satiation. Recently, there has been proof that peripheral CCK may be taken up by the brain to produce its satiety action. The area post Erma contains a deficient blood brain barrier that brings brain tissue in close proximity to the peripheral circulation ***(Bray, 2003)***

6-Other Factors*:*

There is evidence that the size of body fat depots is sensed by either neural or hormonal signals to the brain, and the appetite is controlled in this fashion (lipostatic hypothesis). A cold climate stimulates and a hot environment depresses appetite, Especially in human, cultural factors, environmental, and past experience relative to sight, smell, and taste of food, also affect food intake ***(Kini et al., 200I).***

The sight and smell of food are important signals for initiating food seeking behavior and identifying potential sources of food. Along with the taste and textured food in the mouth, these sensory signals and quality of food can serve both a positive feedback signals, initiating food ingestion, or negative signals to slow down, terminate, or abort any eating incident.obese subjects usually respond to external signals larger than do persons with normal weight ***(Bray, 2002).***

Pathophysiology of Obesity

Energy stored = energy intake - energy expended. So increase the energy intake will lead to more storage of energy in the form of fat ***(Davis, 1998).***

There are several factors concerning the occurrence of obesity the first one in the mechanism is genetic control, central nervous system control, afferent signals, pattern of feeding, socioeconomic factors, exercise and pattern of distribution of excess adipose tissue

1. Genetic control:

Less than 10% of the off spring of lean parents are overweight when one parent is obese, half of the children are obese, and when both parents are obese more than 80% of their children are obese ***(Jensen, 2004).***

Adipose tissue lipoprotein lipase (ATLPL) is an enzyme synthesized within adipocytes secreted into the intercellular space and attached to the luminal border of nearby endothelial cells. In this site (ATLPL) hydrolysis fatty acids from the triglycerides rich lipoproteins, The released fatty acids are taken up by adipocytes, converted to triglycerides and stored thus this enzyme play a major role in the storage of excess fat calories in adipose tissues. In some obese patients excessive levels of this enzyme are detected leading to obesity by excessive deposition of fat in adipose tissue. Also it was found that following weight reduction the level of this enzyme remains high suggesting regaining of lost weight by the obese patients ***(Molina et al., 2003).***

2. Centeral nervous system control:

Hypothalamic inflammatory conditions, trauma or tumors in man cause obesity, more definitely the paraventricular nucleus (PVN) and the ventromedial nucleus (VMN) of the hypothalamus. These nuclei receive messages from the periphery and relay to appropriate sites to initiate or inhibit feeding. Lesions of the paraventricular nucleus produces hyperphagia while lesions of the ventromedial nucleus leading to increased parasympathetic activity leading to increased insulin production, decreased sympathetic activity which reduces thermogenesis and lipolysis, so these effects will lead to obesity ***(Campbell and Haslam, 2005).***

3. Afferent signals:

A) Sensory signals;coming from tast of food when it is delicious.

B) Gastrointestinal signals; Presence of food in gastrointestinal tract send signal in 2 ways; ***1st;*** gastric distension leads to sending stimuli via vagus; ***2nd:*** enterneural axis stimulated by gastro-intestinal hormones that is released by the presence of food in the stomach which stimulate the neural receptors of enteric plexus ***(Campbell and Haslam, 2005)***.

C) Nutrient signals: Increase of the plasma glucose stimulates the satiety center. There is some evidence that obese persons are insensitive to internal stimuli and respond more to external stimuli such as the appearance, aroma and taste of foods. As a result, the obese behavior regarding feeding is dominated by non physiologic external stimuli. So when the latter is removed food intake drops ***(C.Summertan, 2004)***.

4. Pattern of feeding:

The pattern of feeding may influence the balance between energy expenditure and intake. When the daily caloric intake is consumed in large single meal instead of dividing it in three or at dinner time eat a large meal followed by repeated snacking until they retire. More or les common that some obese persons consume the same number of calories as a lean person and yet remain over weight, This is explained by the pattern of feeding or that obese individuals may have more efficient caloric pattern ***(Dietel, 2003).***

5. Socioeconomic factors

These play a secondary but important role. The incidence of obesity is (7-12) times higher in lower socioeconomic groups than among high standard groups. Social factors, advertising in the mass media, occupation and educational level represent additional compounding factors ***(Jung and Cushieri, 2000).***

6. Exercise:

It is the only safe way to increase caloric expenditure. Although it is not proved that reduced physical activity is a major root cause of obesity; obese individuals are relatively inactive. But it is clear that when one increases energy expenditure by physical exercise without increasing caloric intake this weight will be reduced ***(Jensen, 2004).***