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Research Article

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Obesity is Global Health Problem

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Abstract

Overweight and obesity are the result of excessive accumulation of adipose tissue in the body, and due to the spread they represent a global public health problem. Obesity is characterized by the size and number of fat cells. Excess, unnecessary energy is stored by the body in the form of fat stores, in which the human body is much more efficient than in the consumption of fat stores. In highly civilized societies, food is readily available and eating outside the home is common, and the need for physical labor is greatly reduced. The lifestyle has changed significantly in the last century. A sedentary lifestyle dominates our daily lives and most scientists believe that the only culprit for today's obesity pandemic is lifestyle. Man's desire to make his life more comfortable led to a decrease in physical activity and thus energy consumption, while eating habits remained the same, so the diet (daily energy intake) did not adjust to energy consumption.

Keywords: Obesity, Risk, BMI, Diabetes

Introduction

Obesity has reached epidemic proportions worldwide and continues to exact a high cost in human and monetary terms within the United States [1]. This disease is second only to cigarette smoking as a preventable cause of death and deaths attributable to obesity far outnumber colon cancer. Three hundred thousand people die annually from obesityrelated disorders in the United States. In addition, health care costs to treat obesity and weight-related conditions exceed $100 billion annually. This problem is of particular concern because upwards of one-third of adult Americans are obese, with 15% of the population potentially meeting the criteria for bariatric surgery. In short, obesity is a major public health problem that requires aggressive prevention and treatment.

Weight loss surgery has been recognized for decades as an effective treatment of obese individuals. Although the number of bariatric procedures has leveled off over the past few years it is estimated that 340,000 bariatric procedures are still performed annually worldwide. Surgical treatment of obesity is routinely associated with loss of greater than 100 lb. Hence, it is not surprising that patients who undergo such operations can have substantial amelioration of co-morbid conditions.

Astonishingly, on a global scale, 1 billion adults might be classified as overweight, and at least 400 million meet established criteria for obesity [2]. Approximately one third of the adults in the United States are obese, as is at least one in seven children. In the developed world, obesity is more common among women and the poor, whereas in developing countries, it affects primarily the prosperous. The rapid increase in obesity rates indicates that the problem is predominantly environmental and behavioral. Weight gain other than water occurs when energy intake exceeds expenditure: you are what you eat, minus what you burn. Many populations consume an overabundance of calories, and the normal human gastrointestinal tract absorbs essentially all simple fuels presented to it. As little as 300 extra calories per day can result in 120 kg of weight gain from age 15 to age 25. Diabetes is strongly associated with obesity: more than 80% of cases of type 2 diabetes mellitus (T2DM) can be attributed to obesity.

Risk

The fundamental cause of obesity is an energy imbalance between calories consumed and calories expended [3]. Globally, there has been an increased intake of energy-dense foods that are high in fat and a decrease in physical activity due to the modern sedentary nature of many forms of work, changing modes of transportation, and increasing urbanization. In addition, genetic factors may underlie heterogeneous susceptibility for the extent of weight gain upon overeating, physical activity, and our adipogenic environment.

Changes in dietary and physical activity patterns are often the result of environmental and societal changes associated with development and lack of supportive policies in sectors such as health, agriculture, transport, urban planning, environment, food process-

ing, distribution, marketing, communication, and education. Most likely a complex gene–environment interaction determines the individual risk to develop obesity.

In humans, energy homeostasis is under tight control and a stable body weight is very well defended across challenges including times of hunger and overeating. The tight defense of body weight (loss) suggests the existence of a setpoint for body weight, which can vary substantially among individuals and may also vary across lifetime. Appetite and satiety a regulated by a complex system which controls energy homeostasis. This system integrates central pathways and signals from peripheral organs (e.g., leptin from adipose tissue, gut hormone secretion in response to meals, signals from the gastrointestinal nervous system, nutrients). These signals induce a complex response in the central nervous system specifically in the anorexigenic leptin–melanocortin and the orexigenic NPY–AgRP pathway according to dietary intake and nutrient requirements of the organism. Other factors such as insulin may modify these signaling processes and thereby influence energy balance. A complex homeostatic system serves to defend body weight against critical energy deficits or chronic overnutrition. Several adaptive systems are known to restore the initial body weight under such fluctuations of energy intake and expenditure. This may explain why obese humans exhibit a strong tendency to regain weight after intentional dietary weight reduction. The same tendency to return to initial body weight is observed after experimental overfeeding. The role of energy homeostasis in the development of obesity has been elaborated by previous studies using indirect calorimetry to investigate the contribution of the resting metabolic rate to the risk of obesity.

Bmi

Treatment of overweight and obese individuals is based on the degree of excess body weight and the presence or absence of weight-related conditions [1]. The degree and categorization of excess body weight is routinely based on BMI (body mass index). A patient's BMI is calculated by dividing weight in kilograms by the height in meters squared. This index normalizes weight for a given height and is independent of gender. The BMI is generally considered a better classification scheme of excess body weight than the outdated Metropolitan Life tables, which are gender-dependent and require a rough estimate of body frame size.

A BMI of 18.5–25 kg/m2 is considered "normal." A BMI greater than 25 kg/m2 is considered "overweight" or "obese". Based on these criteria an astounding twothirds of adult Americans are overweight or obese. A person is considered obese when his or her BMI is 30 kg/m2 or higher, and obesity is divided into at least three categories, classes I, II, and III. Bariatric surgeons have defined an additional "super obese" category, which is a BMI greater than 50 kg/m2 . This extra surgical category has been used in clinical studies when analyzing data and correlating outcomes with preoperative weight class.

Bariatric Surgery

The indications for surgical treatment of severe obesity are based on the recommendations of the NIH (National Institutes of Health) Consensus Development Conference on Gastrointestinal Surgery for Severe Obesity [1]. The first criterion that must be met before considering a patient for bariatric surgery is weight as assessed by BMI. Patients with a BMI of 35–39.9 kg/m2 (ie, class II obesity) can be considered for surgical intervention if they have severe weight-related conditions such as diabetes, hypertension, debilitating osteoarthritis, or sleep apnea. Those with a BMI of 40 kg/m2 or greater (ie, class III obesity) may be appropriate candidates for bariatric surgery with or without weight-related comorbid conditions. Approval for surgery based on the preceding BMI criteria assumes there are no contraindications to surgery.

If a patient satisfies the BMI criteria for bariatric surgery, he or she is more fully evaluated. The patient should have an extensive history of previous weight loss attempts prior to seeking surgery. Most patients in this weight category have undergone a wide variety of nonsurgical interventions over several years. There should be no unstable psychological conditions or substance abuse. Many surgeons operate on patients with depression, but decline to operate on those with unstable conditions.

Surgical treatment of obesity began in the early 1950s when several groups proposed shortening the intestinal tract via "bypass" procedures to produce substantial decreases in absorptive area. This proposal was based on the observation that massive small bowel resections for treatment of other pathologic conditions resulted in weight loss followed by weight stabilization. Since that time, more than 30 different surgical techniques have been described for treating obesity. The field underwent a recent revolution with the introduction of minimally invasive laparoscopic techniques to produce surgical weight loss. In general, the surgical approach to obesity treatment is designed to create negative energy balance by (1) reducing caloric absorption by way of a small intestinal bypass; (2) reducing caloric consumption by severely restricting gastric capacity; or (3) producing weight loss through a procedure that combines both malabsorption and restriction of caloric intake. Although restriction and malabsorption are the traditional mechanistic ways to categorize weight loss procedures, several research studies indicate that some bariatric procedures are associated with marked neuroendocrine changes. These neuroendocrine changes, particularly the changes observed in gut hormone profiles, are thought to be a significant cause of weight loss after bariatric surgery.

Bariatric operations as a whole have been increasing, with the increase over the last 5–10 years due almost exclusively to the rise of the sleeve gastrectomy as an option for most patients [4]. This rise is based on the ability of most surgeons to perform the procedure via a minimally invasive technique and the lower rate of subsequent operations – at least in the short term. Sleeve gastrectomy has thus largely supplanted the adjustable gastric band as the bariatric surgery option for the most risk-averse patient. The Roux-en-Y gastric bypass remains the default revisional operation of choice because of its effectiveness in challenging situations, and the Roux-en-Y gastric bypass is considered the procedure of choice for patients with pre-existing gastroesophageal reflux and insulin-dependent diabetes mellitus.

There are three major mechanisms of weight loss for the sleeve gastrectomy. One, sleeve gastrectomy creates the greatest restriction of all bariatric operations; early after the operation, food regurgitation is common for dietary indiscretions, thus limiting intake. Two, resecting the fundus reduces ghrelin levels which leads

to a loss of the sensation of ravenous hunger in sleeve gastrectomy patients. Three, removing the fundus leads to rapid gastric emptying which induces a moderate incretin effect. This is responsible for the antidiabetic effect of the sleeve gastrectomy. Because there is no metabolic consequence of softer high-calorie density food in sleeve gastrectomy patients, weight loss is less than that seen in Roux-en-Y patients, and weight regain is more common.

Sedation and Possible Complications

- Sedation-related complication is the leading factor in endoscopy-related mortality
- Assessment of risk factors for sedation-related complications is paramount
- Opiates and benzodiazepines have a synergistic effect

Conscious sedation is defined as the use of medication to depress the central nervous system without the loss of verbal communication [5]. Sedation is the leading factor in endoscopy-related mortality. Risk factors for complications of sedation include the following:

- Advanced age
- Obesity
- Comorbidities including cirrhosis, cardiac disease, respiratory disease, renal disease
- Prior administration of sedation or opiates
- Known drug allergies
- Low resting oxygen saturations
- Emergency endoscopy

Complications of sedation include over-sedation, paradoxical excitement, respiratory depression, aspiration, cardiac arrhythmias, acute coronary events, hypertension, hypotension, cerebrovascular events, nausea, vomiting, and flushing. Risk factors for complications should warrant discussion of un-sedated endoscopy and, if sedation is undertaken, adjustment of doses should be considered.

Anesthesia

The national epidemic of obesity poses particular problems for surgery and anesthesia [6]. The body mass index (BMI), the ratio of weight (kg)/height (m2), gives an idea of the degree of obesity. Normal BMI is about 21.6 kg/m2, overweight is 25-30 kg/m2, obese is 30-35 kg/m2, and extreme obesity is more than 35 kg/m2. Extreme obesity patients have a variety of perioperative issues and should be evaluated in a PAC. Particular attention should include the upper airway and evaluation of cardiovascular, respiratory, metabolic, and gastrointestinal systems. Abnormal BMI patients have cardiovascular issues with venous access, hypertension, cardiomegaly, decreased left ventricular function, and cor pulmonale, and they have twice the incidence of ischemic heart disease than patients at normal weight. Extreme obesity is associated with significant pulmonary problems, including restrictive lung volumes, obstructive sleep apnea, hypoxemia, increased Paco2, increased hematocrit, and right heart failure. The extremely obese patient's airway is often difficult to maintain with mask ventilation secondary to decreased neck mobility and adiposity and requires careful preoperative evaluation. Almost all the major endocrine problems with extreme obesity involve the effects of diabetes mellitus and require preoperative assessment of glycemic control. Obesity also leads to abnormal fatty deposits in the liver that cause increased metabolism of inhalation anesthetics. Morbidly obese patients may have a higher risk of gastric aspiration and development of aspiration pneumonia. Finally, postoperative pain management must be considered.

Diabetes

The liver is adversely affected by several of the metabolic derangements of both type 1 and type 2 diabetes mellitus [7]. Episodic hyperglycemia, increased fatty acid delivery from peripheral stores in times of hypoglycemia, hyperinsulinism in patients with type 2 diabetes, and insufficient insulin in patients with type 1 diabetes all participate in the hepatic complications. The accumulation of fat in the liver of the patient with type 1 diabetes reflects poor glucose control. During periods of insulin deficiency, peripheral fat stores are mobilized, and the free fatty acids released into the circulation are taken up by the liver for conversion to alternative energy forms (eg, ketone bodies). Fatty acids that do not undergo mitochondrial b-oxidation in the liver are recycled back into triglycerides and transported to the peripheral stores by very low density lipoproteins. Any interference in the complex process of synthesizing and secreting triglycerides from hepatocytes is manifested histologically as steatosis. In patients with type 2 diabetes, obesity is the primary cause of fatty liver due to ongoing oversupply of the liver with fatty acids from peripheral stores because of peripheral insulin resistance. Although the accumulation of fat in the liver can cause biochemical evidence of liver injury, diabetes itself rarely, if ever, is a cause of cirrhosis. Impaired liver function caused by other factors (eg, chronic viral or alcoholic cirrhosis) can unmask latent diabetes by increasing peripheral insulin resistance. Thus, an overrepresentation of diabetes in patients with cirrhosis cannot be interpreted as the former causing the latter.

As the prevalence of obesity increases in the American population, so does the prevalence of type 2 diabetes [8]. Ninety percent of all new cases of diabetes diagnosed in the United States are type 2, and it is estimated that this disease affects approximately 7% of the population older than 45 years. Diabetes is the leading cause of blindness, renal failure, and nontraumatic amputations of the lower extremities. It is a major risk factor in patients with coronary artery disease, peripheral vascular disease, and stroke.

In contrast to type 1 diabetics, patients with type 2 diabetes usually have a prolonged asymptomatic phase. During these years of asymptomatic hyperglycemia, however, organ damage begins to occur. Therefore, several organizations recommend screening of certain high-risk populations. The risk factors for diabetes include obesity or overweight (BMI >25 kg/m2); other signs of an insulin-resistance syndrome or "metabolic" syndrome, such as hypertension or low high-density lipoproteins (HDLs) and triglycerides more than 250 mg/dL; first-degree relative with diabetes; history of gestational diabetes; or being a member of a high-risk ethnic group, including African Americans, Hispanics, American Indians, Asian Americans, or Pacific Islanders. Screening should be performed every 3 years beginning at age 45 years, or earlier if overweight with BMI >25 kg/m2.

Most patients with type 2 diabetes mellitus are insulin resistant and

hyperinsulinemic for years before developing overt diabetes. They are able to maintain normoglycemia for a long time, then develop postprandial hyperglycemia, and later develop both postprandial and fasting hyperglycemia (ie, hyperglycemia all the time). Thus, a glucose tolerance test to detect postprandial hyperglycemia would be the most sensitive test for diabetes mellitus but is time consuming and difficult to perform in a clinical practice. The fasting plasma glucose is the most specific test. Hemoglobin A1C (A1C) >6.5% has now also been recognized as an acceptable diagnostic criteria. If there are no clear symptoms of hyperglycemia, the diagnosis of diabetes must be confirmed on a subsequent day by repeat measurement, repeating the same test for confirmation.

Cad

Coronary artery disease (CAD) can cause inadequate myocardial perfusion and poor contractility, resulting in deficient cardiac output and potential heart failure [9]. CAD is the leading cause of death worldwide, and by 2020, it is estimated that CAD will be the leading cause of disease burden (e.g., direct and indirect financial cost, disability, mortality, morbidity) worldwide. In the United States, specifically, CAD places the most severe clinical and financial burden of the healthcare system than any other disease conditions. Currently, over 16 million Americans have CAD, which is the leading cause of cardiovascular death in the United States (one out of every six deaths is caused by CAD). CAD is closely related to other conditions such as obesity, diabetes mellitus, hypertension, and heart failure. As a result, treatment for CAD in the United States leads to the highest cost of any disease condition (~ $100B per year). Between 2010 and 2030, the total direct medical cost of cardiovascular diseases is projected to triple, from $270 billion in 2010 to $800B in 2030.

The coronary circulation consists of an integrated system of complex anatomy, mechanical properties, boundary conditions representing the hemodynamics, and myocardial-vessel interaction, which leads to phasic patterns of coronary blood flow into, within, and out of the myocardium. Coronary blood flow is substantially heterogeneous spatially (throughout the myocardium) and temporally (within cardiac cycle). These temporal and spatial heterogeneities are important physiologically and clinically but are difficult to study at the inner layers of the myocardium, where susceptibility to ischemia is an important clinical phenomenon. Hence, rigorous validated models of the coronary vasculature, mechanical properties, boundary conditions, and myocardial-vessel interaction are critical to produce realistic predictions of blood flow throughout the wall of the heart.

Dietary Fats

Dietary fats are composed primarily of triglycerides (i.e., a compound consisting of three fatty acid chains attached to a glycerol backbone) [10]. Saturated fatty acids have no double bonds between carbon atoms in the chains, whereas monounsaturated fatty acids have one double bond in the chain, and polyunsaturated fatty acids have two or more double bonds. The saturated fatty acids elevate blood cholesterol, whereas the monounsaturated and polyunsaturated fats lower blood cholesterol. Saturated fats are usually derived from animal sources and remain solid at room temperature. With the exception of coconut and palm oils (which are saturated), unsaturated fats are found in plant oils and usually are liquid at room temperature. Trans fatty acids are produced when unsaturated oils are partially hydrogenated and are called artificial trans fats. They are found primarily in vegetable shortenings and some margarines and foods. Small amounts of natural trans fatty acids are found in dairy products, some meats, and other animalbased foods. Although trans fatty acids tend to increase low-density lipoprotein (LDL) cholesterol and decrease high-density lipoprotein (HDL) cholesterol, the naturally occurring trans fats may have a beneficial effect.

Dietary fats provide energy, function as carriers for the fat-soluble vitamins, serve as precursors of prostaglandins, and are a source of the essential fatty acids, linoleic and alpha-linolenic acids. Because vegetable oils are rich sources of linoleic acid, this level can be met by including two teaspoons per day of vegetable oil in the diet. Alpha-linolenic acid is found primarily in dark green, leafy vegetables, certain plant oils, soybeans, and walnuts.

Conclusion

The cause of obesity is not sufficiently explained, and it is an indisputable fact that obesity is a consequence of disproportionate energy intake through food and its consumption in the body with a detrimental predominance of fat deposition in adipose tissue. Two essential sensations, hunger and satiety, are complexly regulated through the center of the sensations of hunger and satiety located in a part of the brain called the hypothalamus. The act of feeding itself is controlled from these centers, which involves stimuli from higher centers in the brain, stimuli from the digestive system, nutrients in the circulation, and some hormones. Total adipose tissue mass can affect the centers in the hypothalamus that seek to keep existing body weight stable.

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