OBSTETRIC PERFORMANCE IN OBESE EGYPTIANS

THESIS

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BY

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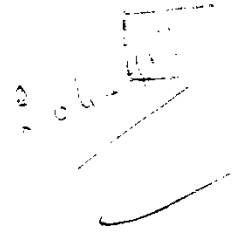

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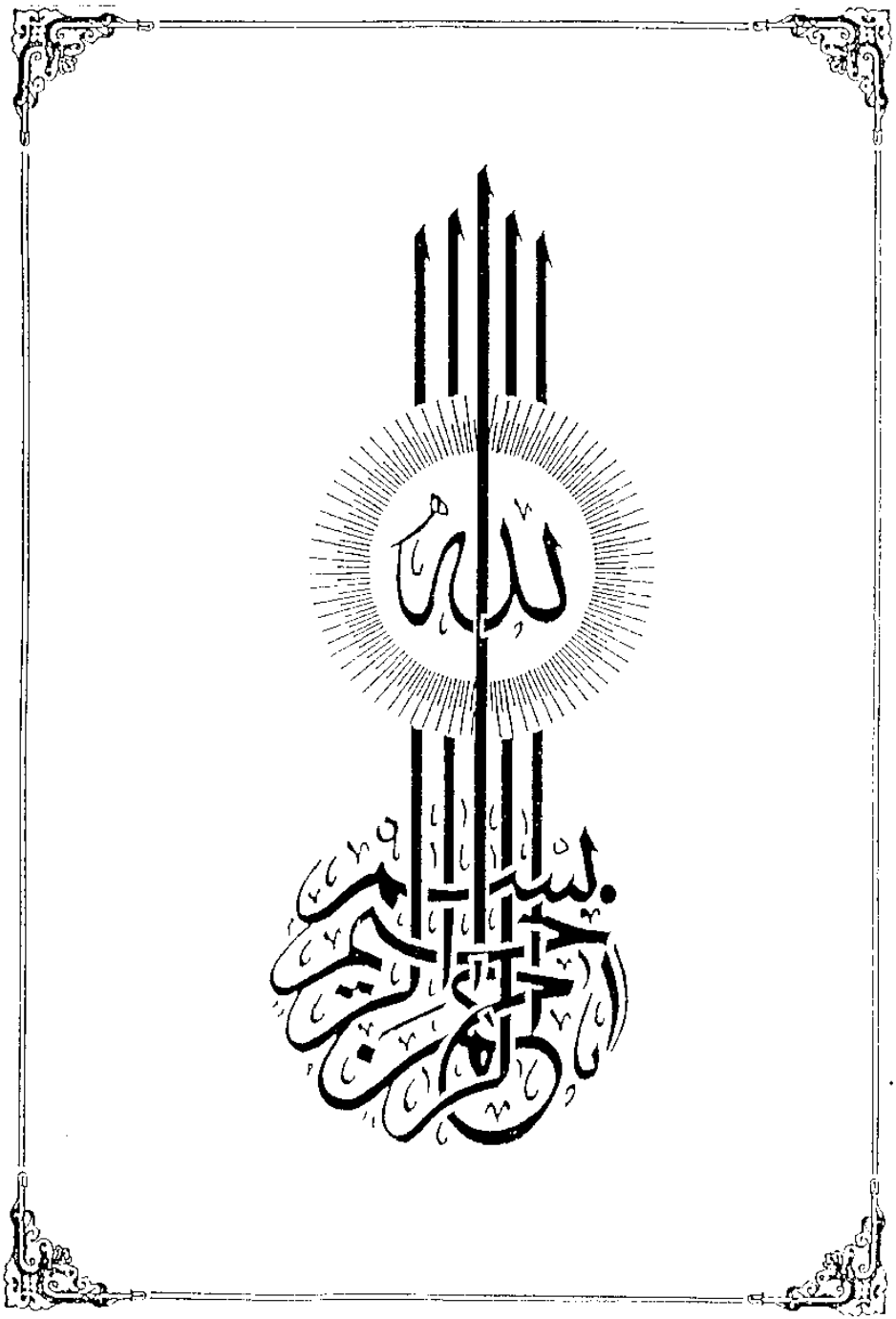

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INTRODUCTION AND AIM OF THE WORK

Obesity is the most common disorder of metabolism in man and is also one of the oldest documented metabolic distrubances in recorded history (Bierman, 1979). A limestone statuette dating from the Stone Age has been unearthed revealing the most ancient example of obesity, antedating the development of agriculture by about 10000 years. Similar historical evidence for obesity is found in Egyptian mummies and Greek sculpture. This abnormality has persisted throughout the centuries, which have been characterized by markedly different environmental stresses and dietary habits. However, the problem of obesity has dramatically increased since the evolutionary advantage of the ability to efficiently store energy as fat has been dissipated in modern affluent societies. Thus caloric excess and sedentary habits have led to an increased prevalence of obesity and its life-shortening consequences, including cardiovascular disease, diabetes, and hypertension (Bierman, 1979).

Obesity is probably the most common problem facing physicians in their everyday practice. The obese parturient, by her weight alone, presents an obstetric risk. In this

work, the aim is to study the effect of obesity on pregnancy, labour, and maternal morbidity on one hand, and its effect on fetal weight, fetal morbidity, and perinatal mortality on the other.

REVIEW OF THE LITERATURE

DEFINITION AND MEASUREMENT

There is a difference between obesity and overweight (Powers, 1980). Obesity is an excess of body fat. Overweight is a body weight in excess of some standard or ideal weight. The ideal weight for any adult is believed to correspond to his or her ideal weight from age 20 to 30.

The following formulas give ideal weight in pounds:

Women: 100 + [4X(height in inches minus 60)]

Men : 120 + [4X(height in inches minus 60)]

At a weight close to ideal weight, individuals may be overweight, but not overfat.This is especially true of individuals engaged in regular exercise. An estimate of body fat, therefore, rather than a measurement of height and weight, is more significant.

The most accurate method of determining body fat is to determine the density of the body by underwater

measurement. It certainly is not practical to measure density by submerging individuals in water in our offices, therefore skinfold measurements with calipers have become popular as an index of body fat. The skinfold measurement is also not necessary for clinical practice. It is far simpler to utilize the body mass index nomogram, a method which has been found to correspond closely to densitometry measurements (Thomas et al., 1976).

The body mass index is the ratio of weight divided by the height squared (in metric units). To read the central scale in Figure (1) align a straight edge between height and body weight. A body mass index of about 30 is roughly equivalent to 30% excess body weight, the point at which excess mortality begins. Above 40, the risk from obesity itself is comparable to that associated with major health problems such as hypertension and heavy smoking.

A person is obese when the amount of adipose tissue is sufficiently high (20% or more over ideal weight) to detrimentally alter biochemical and physiologic functions and to shorten life expectancy. Obesity is associated with four major risk factors for atherosclerosis: hypertension, diabetes, hypercholesterolemia, and hypertriglyceridemia. Overweight individuals have a higher

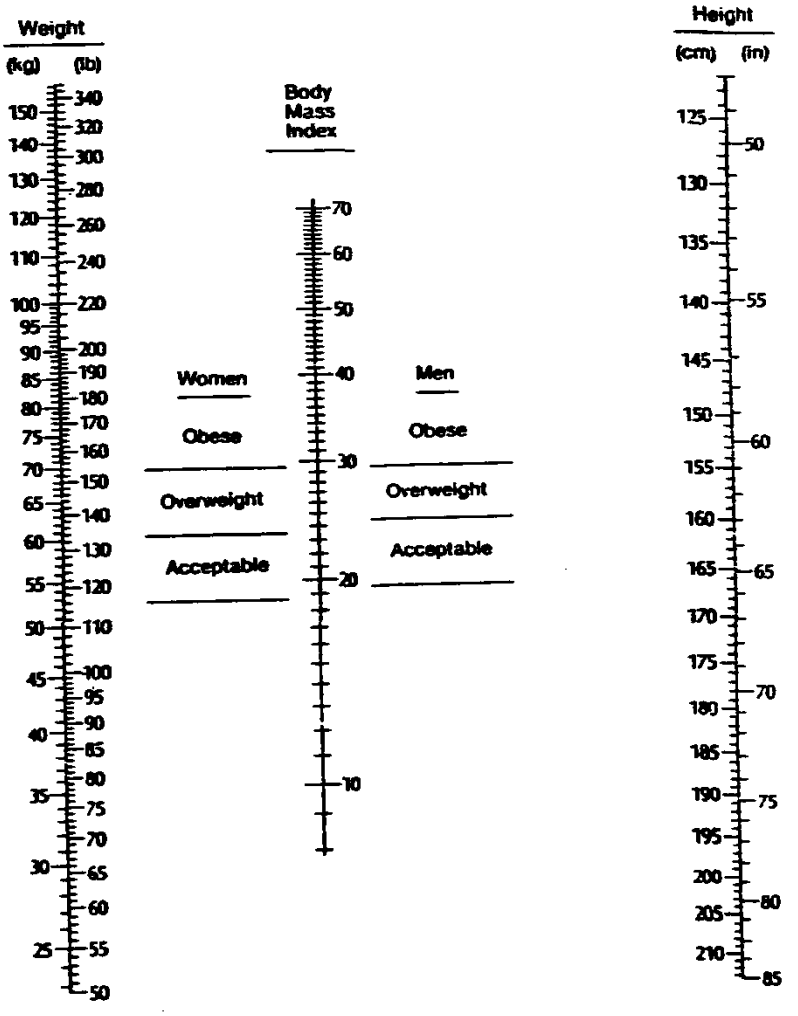

Fig. (1)

prevalence of hypertension at every age, and the risk of developing hypertension is related to the amount of weight gain after age 25 (Stamler et al., 1978). The two in combination (hypertension and obesity) increase the risk of heart disease, cerebrovascular disease, and death.

Unfortunately the basal metabolic rate decreases with age. A 30 year old individual will inevitably gain weight if there is no change in caloric intake or exercise level over the years. The middle aged spread is both a biological and a psychosocial phenomenon. It is therefore important for both our patients and ourselves to understand adipose tissue and the problem of obesity.

PHYSIOLOGY OF ADIPOSE TISSUE

Adipose tissue serves three general functions:

1. Adipose tissue is a storehouse of energy.
2. Fat serves as a cushion from trauma.
3. Adipose tissue plays a role in the regulation of body heat.

Each cell of adipose tissue may be regarded as a package of triglyceride, the most concentrated form of stored energy. There are 8 calories per gram of triglyceride as opposed to 1 calorie per gram of glycogen. The total store of tissue and fluid carbohydrate in adults (about 300 calories) is inadequate to meet between-meal demands. The storage of energy in fat tissue allows us to do other things besides eating. The mechanism of mobilizing energy from fat involves various enzymes and neurohormonal agents. Following ingestion of fat and its breakdown by gastric and pancreatic lipases, absorption of long-chain triglycerides and free fatty acids takes place in the small bowel. Chylomicrons (microscopic particles of fat) transferred through lymph channels into the systemic venous circulation are normally removed by hepatic parenchymal cells where a new lipoprotein is released

into the circulation. When this lipoprotein is exposed to adipose tissue, lipolysis takes place through the action of lipoprotein lipase, an enzyme derived from the fat cells themselves. The fatty acids that are released then enter the fat cells where they are reesterified with glycerophosphate into triglycerides (Speroff et al., 1983).

Glucose serves three important functions:

1. Glucose supplies carbon atoms in the form of acetyl coenzyme A (acetyl CoA).
2. Glucose provides hydrogen for reductive steps.
3. Glucose is the main source of glycerophosphate.

The production and availability of glycerophosphate (required for reesterification of fatty acids and their storage as triglycerides) are considered rate-limiting in lipogenesis, and this process depends on the presence of glucose.

After esterification, subsequent lipolysis results in the release of fatty acids and glycerol. In the cycle of lipolysis and reesterification, energy is freed as heat. A low variable level of lipolysis takes place continuously; its basic function may be to provide body heat.

The chief metabolic products produced from fat are the circulating free fatty acids. Their availability is controlled by adipose tissue cells. When carbohydrate is in short supply, a flood of free fatty acids can be released. The free faty acids in the peripheral circulation are almost wholly derived from endogenous triglyceride that undergoes rapid hydrolysis to yield free fatty acid and glycerol. The glycerol is returned to the liver for resynthesis of glycogen.

Free fatty acid release from adipose tissue is stimulated by physical exercise, fasting, exposure to cold, nervous tension, and anxiety. The release of fatty acids by lipolysis varies from one anatomic site to another. Omental, mesenteric, and subcutaneous fat are more labile and easily mobilized than fat from other sources. Areas from which energy is not easily mobilized are retrobulbar and perirenal fat where the tissue serves a structural function. Adipose tissue lipase is sensitive to stimulation by both epinephrine and norepinephrine. Other hormones that activate lipase are ACTH, thyroid stimulating hormone (TSH), growth homrone, thyroxine(T_4), 3,5,3'-triiodothyronine (T_3), cortisol, glucagon, as well as vasopressin and human placental lactogen (HPL) (Speroff et al., 1983).

Lipase enzyme activity is inhibited by insulin, which appears to be alone as the major physiologic antagonist to the array of stimulating agents. When both glucose and insulin are abundant, transport of glucose into fat cells is high, and glycerophosphate production increases to esterify fatty acids.

The carbohydrate and fat composition of the fuel supply is constantly changing, depending upon stresses and demands. Since the central nervous system and some other tissues can utilize only glucose for energy, a homeostatic mechanism for conserving carbohydrate is essential. When glucose is abundant and easily available, it is utilized in adipose tissue for producing glycerophosphate to immobilize fatty acids as triglycerides. The circulating level of free fatty acids in muscle will, therefore, be low, and glucose will be used by all of the tissues.

When carbohydrate is scrace, the amount of glucose reaching the fat cells declines and glycerophosphate production is reduced. The fat cell releases fatty acids, and their circulating levels rise to a point where glycolysis is inhibited. Thus, carbohydrate is spared in

those tissues capable of using lipid substrates. If the rise of fatty acids is great enough, the liver is flooded with acetyl CoA. This is converted into ketone bodies, and clinical ketosis results.

In the simplest terms, when a person eats, glucose is available, insulin is secreted, and fat is stored. In starvation, the glucose level falls, insulin secretion decreases, and fat is mobilized (Speroff et al., 1983).

If only single large meals are consumed, the body learns to convert carbohydrate to fat very quickly. Epidemiologic studies on school children demonstrate a positive correlation between fewer meals and a greater tendency towards obesity (Fabry et al., 1966). The person who does not eat all day and then stocks up at night is perhaps doing the worst possible thing.