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Obesity: «Οὐκ ἐν τῷ Πολλῷ τὸ Εὐ» (Less is More)

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Obesity is characterised by an excessive deposition of fatty tissue in the body and those who suffer from it experience physical, psychological, and social consequences. Although it was officially recognised as a disease in 1948, when the World Health Organisation was founded and included it in the International Classification of Diseases, its history goes back a good deal further.[1]

Dating back to the Palaeolithic era, many idols have depicted goddesses with obese bellies, outsize breasts, and fat buttocks. Similar forms appear in statuettes from Mesopotamia, as well as in Pharaonic mummies and wall paintings in Egyptian tombs. There are similar reports in Chinese, Tibetan, and Indian medicine, as well as in the medicine of the Mayans and Aztecs, thus demonstrating that obesity had made its presence felt even in the distant past.

In his writings, Hippocrates, the father of medicine, maintained that "sudden death is more common in obese people than in thin people." Galen classified obesity as moderate or excessive, where the former was considered normal and the latter unhealthy. From Roman times, too, there are descriptions associating obesity with somnolence and sleep apnoea, and cases were reported of people who died from excessive weight. The common point of reference of all the therapeutic approaches to the treatment of obesity in ancient times involved diet and exercise. Hippocrates, Galen, and much later Avicenna, all recommended the same things: vigorous exercise, a frugal diet, and abstemiousness.

Unfortunately, after 2500 years Hippocrates and his successors have still not managed to persuade us that obesity is a disease, and a very dangerous one. Thus, today we find ourselves in the midst of an obesity pandemic, extending beyond the Western world, and affecting not only adults, but children and teenagers. For the first time in humanity's history, our planet is "host" to more obese and overweight people than those who suffer from malnutrition. Based on the latest WHO data, worldwide there are about 2 billion people who are overweight and more than 700 million who are obese. In Greece, obesity is a major public health problem, as Greek children are the most obese children in Europe, while adults are not far behind. In this country, 35.2% of the general population are overweight, while 22.5% are obese.[2]

The rapid increase in the prevalence of obesity during the last 50 years is the result of our evolutionary development. The progressive improvement in living conditions has led to increased food consumption and a sedentary lifestyle, culminating in a positive energy balance and an increase in bodily fat. Indeed, an absence of physical activity and a sedentary lifestyle are basic characteristics of the modern way of life. Studies have shown that adults spend more than half their time at work sitting down. Similarly, the greater part of their home activities involves watching television and surfing the Internet.

There is a wealth of persuasive findings to show that obese people are at increased risk for a series of health problems, including type II diabetes mellitus, arterial hypertension, dyslipidaemia, coronary artery disease, stroke, osteoarthritis, sleep apnoea syndrome, and certain forms of cancer—conditions that affect the quality of life and clearly shorten life expectancy—compared to individuals of normal weight. It is estimated that 44% of the burden of diabetes, 23% of the burden of ischaemic heart disease, and between 7% and 41% of the burden of certain types of cancer, can be attributed to increased body weight and obesity. Recent findings show that obesity is associated with a greater morbidity than smoking, alcohol, and poverty, and that if current trends continue,

obesity will soon overtake smoking as the leading reversible cause of death in the USA.[3]

The correlation between obesity and various types of cardiovascular disease is complex, probably because of the different pathophysiological mechanisms that involve a large number of interacting factors. Obesity may cause coronary atherosclerosis via well studied and widely accepted mechanisms, such as dyslipidaemia, arterial hypertension, and type II diabetes mellitus. However, recent findings have shown that the correlation between obesity and cardiovascular diseases can be influenced by many other factors, such as subclinical inflammation, neurohormonal activation with an increase in sympathetic tone, high concentrations of leptin and insulin, obstructive sleep apnoea, and an increase in the circulation of free fatty acids.[4]

The increased abdominal fat that accompanies central obesity is the most active fatty tissue and causes increased insulin resistance, elevated triglycerides, an alteration in the magnitude of low-density lipoproteins (LDL) and a reduction in high-density lipoproteins (HDL). The mechanisms via which the increased fat deposition causes insulin resistance are complex. Various pathophysiological pathways are involved, via the intervention of cytokines, other inflammatory agents, and increased leptin levels.

Leptin is an important peptide hormone that plays a fundamental role in the feeling of satiation and in the energy balance. Obese individuals show greatly elevated levels, because of leptin resistance. However, endogenous hyperleptinaemia does not reduce the appetite and does not increase energy consumption. Rather, it increases sympathetic activity, promoting thrombosis, together with an increase in blood pressure and heart rate. Leptin is a cytokine and is therefore involved in the inflammatory process. The voluntary loss of weight, especially the reduction in fatty tissue, leads to a reduction in circulating leptin levels.

In addition, obesity disturbs the structural and functional properties of the cardiovascular system, because of an increase in total blood volume and cardiac output, with the result that the cardiac load is greater in obese patients. Typically, the obese have a higher cardiac output, but lower overall peripheral vascular resistances, for any given level of blood pressure. The greater part of the increase in cardiac output is caused by the increase in stroke volume, even though, because of the enhanced sympathetic stimulation, the heart rate is also increased. The Frank–Starling curve is often displaced towards the left, because of an increase in pressure and filling volume, and this leads to an increase in cardiac work. Obese patients are more likely to be hypertensive compared to individuals of normal weight, and an increase in body weight is associated with a further increase in blood pressure.

With the increase in pressure and filling volume, overweight and obese individuals often develop left ventricular dilation. Even independently of blood pressure and age, obesity increases the risk of left ventricular hypertrophy and causes disturbances of left ventricular geometry, characterised by concentric remodelling and concentric hypertrophy. In addition to the structural effects on the left ventricle, obesity leads to dilation of the left atrium, because of both the increase in circulating blood volume and the impaired left ventricular filling. Not only do these disturbances increase the risk of heart failure, but the left atrial dilation can increase the risk of atrial fibrillation. The structural and functional disturbances of the left ventricle make it more prone to complex ventricular arrhythmias and have a negative effect on its systolic and diastolic performance.

Paradoxically, while obesity is associated with increased mortality and morbidity in the general population, in specific groups of patients (e.g. those with atrial fibrillation, heart failure, coronary artery disease, peripheral artery disease, cerebrovascular disease, cancer, diabetes mellitus, chronic obstructive pulmonary disease, HIV/AIDS, and the elderly) it offers a survival benefit. This finding has been confirmed in large series of patients and in meta-analyses; it is referred to in the literature as "the obesity paradox", and it has not yet been adequately explained. However, recent data point to a good natural cardiopulmonary condition as a powerful modifier of the risk of obesity.

Indeed, there are persuasive findings that support the benefits of regular physical exercise, healthy diet, and the maintenance of an ideal body weight in reducing cardiovascular morbidity and mortality, as well as improving the quality of life.[5]

Today, in contrast to the past, the treatment of obesity is inextricably linked with prevention. It is clear that in ancient times, doctors and all kinds of scientific healers attempted to deal with the phenomenon when it was already apparent. There are no indications of them having given instructions or advice not to become obese. Nowadays, this has become something more than necessary. Prevention strate-

gies in the general population are based mainly on efforts to educate and inform citizens comprehensively regarding matters of diet and physical activity. More than anything, all citizens should be made aware that obesity is a chronic disease with severe complications, which is more easily prevented than treated.

Furthermore, there is a pressing need to guide those suffering from obesity to maintain healthy dietary and exercise habits. The measurement of body mass index and waist circumference are basic tools for determining the degree and distribution of obesity, and can be used for the determination of patients' cardiovascular and metabolic risk profile.[6]

Significant progress has been made with regard to dietary, exercise, pharmaceutical, and bariatric surgical approaches to the successful long-term treatment of obesity. Lifestyle and dietary interventions aimed at reducing energy intake and increasing energy consumption, via a balanced diet and exercise programme, remain the cornerstones in the treatment of obesity. Dietary recommendations should include a reduction in the consumption of fats and sugar and an increase in consumption of fruit and vegetables. Exercise recommendations should include 30 to 60 minutes of daily aerobic exercise of moderate intensity (4-6 METS), in combination with two sessions of muscle strengthening per week. In selected patients, such as asymptomatic individuals with high cardiovascular risk, symptomatic patients, and patients with known cardiovascular disease, an exercise stress test should be performed before they begin participation in exercise programmes. A negative stress test and the ability to exercise to above 5 METS mean that the patient is able to start a daily exercise programme of progressively increasing intensity. Patients with cardiovascular disease and evidence of a high risk can also benefit from exercise programmes, but only under close medical supervision. In selected patients with comorbidities, in whom the above measures are unsuccessful in reducing body weight, the addition of pharmaceutical and surgical interventions can lead us safely to the desired result.[7]

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