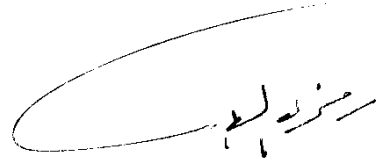


OBESITY IN CHILDHOOD

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An Essay

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BY

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TO

MY

WIFE , MY DAUGHTER AND MY SON
FOR THEIR SUPPORT , PATIENCE , AND
UNDERSTANDING.



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Introduction

INTRODUCTION AND AIM OF THE ESSAY

Obesity is a prevalent condition in childhood and is associated with an increased risk of becoming an obese adult. The importance of treating the obese child and attempting to prevent this child from becoming an obese adult is based in part on the observation that treatment of obesity in adults has not been very successful, obese adults lose a relatively small amount of their excess weight in most treatment program (Wing and Jeffery , 1979).

Thus, it is not advantageous to wait until an obese child becomes an adult and then attempt to achieve ideal weight. In addition, childhood obesity is related to many of the same cardiovascular risk factors as is adult obesity. Obese children have elevated blood pressures and elevated total cholesterol relative to thinner children. These early elevations may be important because these risk factors track over time so that children with elevated blood pressures or lipids may become adults with hypertension or hyperlipidemia (Epstein et al., 1985).

Most atherogenic serum lipid disorders appear to have their origin in childhood. (Kannel and Dawber, 1972).

The tendency for obese children to remain obese as adults links childhood obesity to some of the most

serious disorders of adult life - diabetes, hypertension, and coronary heart disease. This tendency is a strong one indeed. More than 80 percent of overweight children become overweight adults (Brownell and Stunkard, 1980).

Obese children and adolescents are frequently among the most difficult and frustrating management problems confronted by pediatricians. A small fraction of such patients will have a rare genetic or endocrine abnormality, but the majority will have the poorly understood syndrome called, for lack of a better term, "exogenous obesity." The physician needs to be able to eliminate the possibility of endogenous obesity and then to aid the exogenously obese child and his family positively and comfortably. This requires realistic understanding of the physical and emotional risks associated with obesity, alertness to the potential for early intervention, and knowledge of current therapeutic regimens (Golden, 1979).

Obesity is widely recognized as a major health problem of the 20 th century (Johnston, 1985).

It is a subject of universal interest and many studies have been done upon childhood obesity from which an Egyptian study done in 1983 by Abdel - Alim .

The importance of obesity requires constant emphasis because the excess mortality and numerous complications it carries.

Review of Literature

DEFINITION

Obesity is defined as an excessive accumulation of fat when body weight exceeds the normal by at least 20 percent (Simic,1980) and (Vasselli et al,1983).

The most widely used method of defining obesity has been by relating body weight to chronological age. A mild refinement is to consider weight as it relates to length. Obviously a combination of both length and age adds a little more refinement to the above concept (Mc Laren and Read,1972).

Dr Shukla and others in 1972 defined individuals as overweight if their relative weight/height for age was 10 % greater than the expected standard , and assumed that infants who were 20 % over the expected standards were obese.

The relative weight = Body weight divided by the midpoint of desirable weight (Consensus Conference 1985).

Jung in (1985)suggested that "Overweight" describes a weight which in 110 - 119 % above the upper limit of an "acceptable" weight range and "Obesity" describes a weight of 120 % or above. He added that obesity is often expressed in terms of the body mass index (BMI). A body

mass index of 30 or more indicates obesity.

Also Baird(1984)reported that A body mass index (W/H^2) or (Kg/m^2) of more than 25 is also indicates obesity.

Infants whose weight exceeds two standard deviations for age are considered to be overweight but may or may not be obese. And the infant whose weight is in excess of three standard deviations and whose length is not proportionally increased is much more likely to be obese (Weil,1975).

Mayer(1966)suggested that triceps skin fold thickness greater than 15 mm in males and 17 mm in females should be defined as obesity.

Obesity is best defined as an excessive accumulation of triglyceride fat in the adipose tissue depots of the body (Anderson, 1972).

PHYSIOLOGY OF APPETITE AND CONTROL OF FEEDING

Animal studies have shown the presence of hypothalamic appetite and satiety centres, and the impression in man is that these may be influenced by nervous or humoral pathways from the stomach and duodenum. (Anderson, 1972).

Feeding and Satiety Centers :

Hypothalamic regulation of the appetite for food depends primarily upon the interaction of 2 areas : a lateral "feeding center", and a medial "satiety center" in the ventromedial nucleus. Stimulation of the feeding center evokes eating behaviour in conscious animals, and its destruction causes severe, fatal anorexia in otherwise healthy animals. Stimulation of the ventromedial nucleus causes cessation of eating, whereas lesions in this region cause hyperphagia and, if the food supply is abundant, the syndrome of hypothalamic obesity occurs.

Destruction of the feeding center in rats with lesions of the satiety center causes anorexia, which indicates that the satiety center functions by inhibiting the feeding center.

The feeding center is chronically active and its activity is transiently inhibited by activity in the satiety center after the ingestion of foods (Ganong, 1983).

Anand in(1961)classified the stimuli affecting the centres based on the hypothalamus into chemical, nervous and thermal stimuli.

A. Chemical Stimuli :

The concentration of the glucose and free fatty acid in the blood, there alteration leads to the feeling of hunger (Anand , 1961).

1. Blood glucose :

The activity of the satiety center is probably governed in part by the level of glucose utilization of cells within the center. These cells have therefore been called glucostats. It has been postulated that when their glucose utilization is low- and consequently when the arteriovenous blood glucose differences across them is low - their activity is decreased. Under these conditions, the activity of the feeding center is unchecked, and the individual is hungry. When utilization is high, the activity of the glucostats is increased, the feeding center is inhibited, and the individual feels sated (Ganong , 1983).

2. Serum F.F.A. (nonesterified fatty acids)

This type of fat is transported from the adipose tissue to the muscles to be used as source of energy when serum glucose declines, it was found that the sense of hunger is proportional to the serum F.F.A. concentration (Dole, 1965).

B. Nervous Stimuli :

Some of these stimuli reach the hypothalamus due to hunger pains when the subjects stomach or intestine is empty of food, the ultimate result is stimulation of the "feeding centre".

Other stimuli arise from the adipose tissue to the hypothalamus informing about the condition of the fat cells. (Anand , 1961).

C. Thermal Stimuli :

A cold environment stimulates and a hot environment depresses appetite (Ganong, 1983).

Other Factors Regulating Food Intake :

The limbic system is also involved in the neural regulation of appetite. Lesions of the amygdaloid nuclei