GROWTH HORMONE LEVEL IN OVERWEIGHT CHILDREN

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Pediatrics

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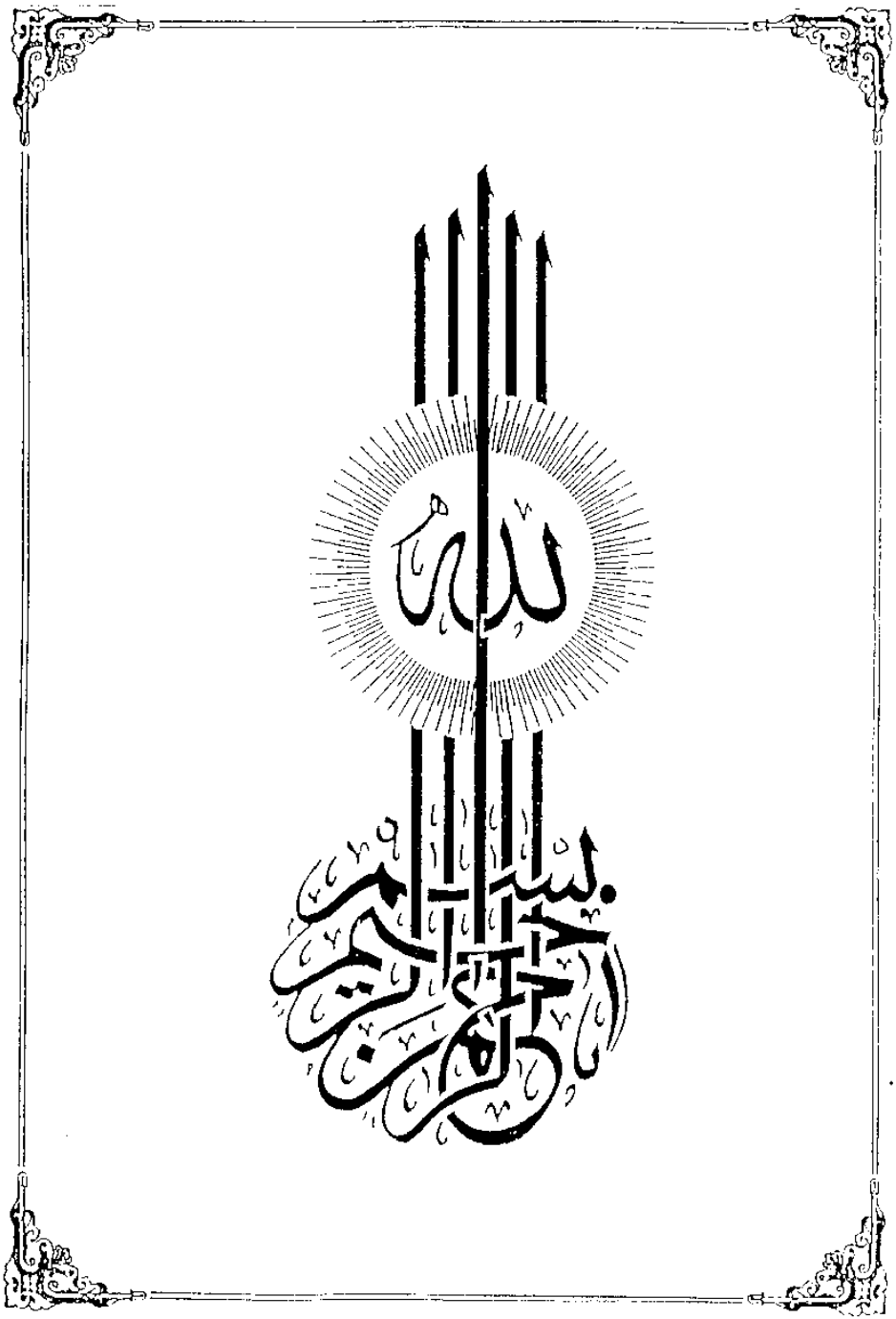

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ABSTRACT

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Adulthood obesity is considered a serious disorder of adult morbidity and as obesity in childhood is usually associated with an increased risk of becoming obese adults, thus this study was carried out to investigate the relation between growth hormone secretion and simple obesity in childhood. Results revealed that cases had low basal growth hormone level, also had blunted response of growth hormone level after clonidine adminstration.

Key words:- Growth hormone - Obesity - Children.

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Abbreviations

VMN	=	Ventromedial nucleus.
hGH	=	Human Growth Hormone.
GH	=	Growth Hormone.
GHRH	=	Growth Hormone Releasing Hormone.
BMI	=	Body Mass Index.
CT	=	Computed Tomography.
MRI	=	Magnetic Resonance Imaging.
US	=	Ultra Sonography.
GHD	=	Growth Hormone Deficiency.
IGF I and II	=	Insulin Like Growth Factor I & II.
GHBP	=	Growth Hormone Binding Protein.
GHIH	=	Growth Hormone Inhibiting Hormone.
Sms	=	Somatomedins.
SmC	=	Somatomedin-C.
PNL	=	Polymorphonuclear Leucocytes.
TSH	=	Thyroid Stimulating Hormone.
TBG	=	Thyroid Binding Globulin.

REVIEW OF LITERATURE

INTRODUCTION

Obesity is defined as weight >20% than that expected for height and sex mainly due to excess subcutaneous fat. In children, simple obesity is characterised by normal or increased growth rate with acceleration of bone maturation, 25% of boys and 29% of girls were above the 90th percentile for height. As these approached adolescence they moved closer to & then below average stature **[Vandershueren, 1993].**

When longitudinal growth slows down in presence of obesity a hormonal disturbance should be sought for. Most of obese subjects are characterised by several neuroendocrine abnormalities **[Pombo, et al., 1989].**

Growth hormone is an anabolic hormone which may effect the amount and distribution of adipose tissue, **[Nishi, et al., 1993] [Araeonit, et al., 1993].**

It is reported that there is reduced growth hormone secretion in obese children and this may be as low as in poorly growing children with classical growth hormone deficiency **[Ghigo, et al., 1992].**

Thus the hormonal disturbance that produces disturbed growth in obesity remains to be elucidated and offers a fertile field for investigation.

Aim of the present study:-

Is to reveal any changes in growth hormone level (basal and after clonidine) between obese and non-obese children. The level of this hormone will be correlated with parameters of growth as height, weight, span, midarm circumference, skull cicumference, fasting blood glucose and X-ray left carpal bone.

whereas lesions in this region cause hyperphagia, and if the food supply is abundant, the syndrome of hypothalmic obesity occur. **(Ganong, 1983)**.

Destruction of the feeding center in rats with lesions of the satiety center causes anorexia which indicates that satiety center functions by inhibiting the feeding center. The feeding center is chronically active and its activity is transiently inhibited by the activity in satiety center after ingestion of food. **(Ganong, 1983)**.

Some physiological factors in the obese phenotype give rise to rapid weight gain and tend to maintain the obese state, e.g. inactive brown fat and lower thermogensis, increased level of the enzyme lipoprotein lipase, also increased adipose cell number, reduced metabolic rate. In effect obesity phenotype. **(Brownell & Forety, 1986)**.

Determination and Measurement of Obesity:-

The normal evolution of adipose tissue comprises several phases during the first year of life adiposity increases rapidly due to the growth in the size of the adipocytes, which diminishes the next year or two and then remains stable for several years. While adipose tissue growth is decreasing, body height continues to increase. The onset of 2nd period of rapid growth in body fat, termed the "Adiposity rebound" starts about 6 years of age, from this moment both the size and number of adipocytes increase **(Cachera et al, 1984)**.

Adiposity increases during the first year of life and then decreases, a renewed rise termed adiposity rebound. There is a study showing a relationship between the age at adiposity rebound and final adiposity. An early rebound (before 5.5 yrs age) is followed by a significantly higher adiposity level than a later rebound (after 7 yrs of age). **(Cachera et al, 1984)**.

Evolution of adiposity, already been determined as follows:-

(1) Obese children at 1 yr with a precocious adiposity rebound will remain obese.

(2) Obese children at 1 yr with late rebound will eventualy join the average group .

(3) non obese children at 1 y with normal or delayed rebound will remain average or low weight.

(4) non-obese children at 1 yr with advanced rebound (before 5.5 yr of age) will reach the higher percentiles, their overweight will be detected several years ofter rebound. **(Cachera et al, 1984).**

Obesity however, how proved diffcult to quantify, Body mass indices (BMIs) are frequently included in epidemiological studies as estimates of body adiposity. A number of body mass indices have been developed, all are derived from body weight and height measurements. The more popular BMIs include the weight height ratio (W/H). Quetlet index W/H^2 and Khosla-lowe index (W/H^3). These indices were widely used in large epidemiologic and health investigations because of their simpilicity and cost effectiveness.

Measurement of obesity :-

I - Standard Height -Weight Tables

II - Weight to Height Ratio.

III - Skin-Fold Thickness.

IV- Waist to Hip Circumference.

V- Dual-Photon Absorptiometry

VI - Other Methods:

1. **Total body density**
2. **Total water estimates**
3. **Total body potassium measurement.**

I- Standard Height - Weight Tables:-

The median of standard values of weight for length or height of boys and girls are represented in Table **(James, 1985).**

Length or Height "Cm"	Median weight "kgm"		Length or Height "Cm"	Median weight "kgm"	
	Boys	Girls		Boys	Girls
50	3.3	3.4	100	15.5	15.2
52	3.7	3.7	102	16.3	15.9
54	4.1	4.1	104	16.8	16.6
56	4.6	4.5	106	17.4	17.0
58	5.1	5.0	108	18.1	17.6
60	5.7	5.5	110	18.7	18.2
62	6.2	6.1	112	19.4	18.9
64	6.8	6.7	114	20.0	19.6
66	7.4	7.3	116	20.7	20.3
68	8.0	7.8	118	21.5	21.0
70	8.5	8.4	120	22.2	21.8
72	9.1	8.9	122	23.0	22.7
74	9.6	9.4	124	23.9	23.6
76	10.0	9.8	126	24.8	24.6
78	10.5	10.2	128	25.7	25.7
80	10.9	10.6	130	26.7	26.8
82	11.3	11.1	132	27.8	28.0
84	11.7	11.4	134	29.0	29.4
86	12.1	11.8	136	30.2	30.8
88	12.5	12.2	138	31.5	
90	13.0	12.6	140	33.0	
92	13.4	13.0	142	34.5	
94	13.9	13.5	144	36.1	
96	14.4	14.2			
98	14.9	14.5			

The median standard values for wewight for length or hieight of boys and girls **(James, 1985).**

Quoted from Oxford textbook of Medicine, 1985.

II- <u>Weight to Height Ratios</u> :-

The most widely used clinical tool for the assessement of obesity is the BMI; defined as weight divided by the square of height (W/H^2), in Kilograms per meter. **(Black et al, 1983).**

Formulas have been derived for calculating percent of fat in the body from the BMI; (1) for men: % of fat = 1.218 (W/H^2)-10.13; (2) for women% fat=1.48 (W/H^2)-7. **(Black et al, 1983).**